JOURNAL of The IOWA MEDICAL SOCIETY

IN THIS ISSUE:

SEPTEMBER 1961


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(1) Carter, S.: *M. Clin. North America* 37:315, 1953.
(2) Maltby, G. L.: *J. Maine M. A.* 48:257, 1957.
(3) Buchthal, F.; Svensmark, O., & Schiller, P. J.: *Arch. Neurol.* 2:624, 1960.

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Scientific Articles

Problems in the Treatment of Carcinoma of the Rectosigmoid and Rectum

RICHARD L. LAWTON, M.D.

IOWA CITY

THE PROBLEMS associated with the treatment of carcinomas of the rectosigmoid colon and rectum relate primarily to the control of local lesions and their contiguous and distant spread. Important considerations include choice of operative procedure, extended operations, limited resection in the poor-risk patient, palliative procedures and the management of superimposed inflammation.

Local recurrences often indicate technical failure, and it is in this area that refinements in surgical technics can increase the cure rate. We usually accept distant spread as something beyond our control at the present time. The presence of contiguous spread does not negate the possibility of cure, but it does necessitate devising operative procedures of a more radical nature.

Excisional surgery has just about reached its apogee. This prompts us to seek refinements in surgical technic and to look for adjuvant measures. The accent at this time, insofar as adjuvant measures are concerned, is on cancer chemotherapy.

Patients with untreated carcinoma of the colon and rectum survive approximately 17 months. Nearly 100 per cent of these patients are operable by present-day standards. Of this group, 90 per cent are resectable. The operative mortality varies from 3 to 8 per cent. The absolute five-year survival rate is about 25 per cent, and the relative five-year cure rate is 60 per cent. The cure rate with positive nodes present is approximately 30 per cent. Twelve per cent of the patients operated upon have hepatic metastases that are palpable or visible at operation. Some patients operated upon will have hepatic metastases that are not detected at the time of operation.

Dr. Lawton is a member of the surgical staffs at the Veterans Administration Hospital in Iowa City and the S.U.I. College of Medicine. He made this presentation at the Postgraduate Conference on Surgery held on December 6 and 7, 1960, in Iowa City.

ANATOMY

The anatomy of the lymphatics is of particular importance to surgeons. It has been demonstrated that the lymphatic channels and nodes generally parallel major arterial vascular channels. In the rectosigmoid and rectum, we are particularly interested in the vascular axis of the inferior mesenteric artery, the middle hemorrhoidal artery and the hypogastric complex. It has been demonstrated that 8 per cent of metastases from cancer of the rectosigmoid and rectum will be found near the root of the inferior mesenteric artery, and that about 22 per cent of metastases will be found within the first 5 cm. from the root of the vessel.[1] Studies of the spread of tumor in lymphatic channels and to nodes distal to the tumor have demonstrated that distal spread is present to any great degree only in late cases.

For purposes of orientation, something must be said about distances as they relate to the rectum and rectosigmoid. It should be remembered, however, that there is great variation in distances as measured in this region. The point of reference frequently used is the anal verge. This has been variously defined as the apparent external opening of the anal canal, or more specifically the junction of the hairless anoderm with the hair-bearing perianal skin. The distance from the anal verge to the peritoneal reflexion is between 7 and 9 cm. The peritoneal reflexion is here defined as the area where the peritoneum leaves the side of the rectum and proceeds to the lateral pelvic walls. This can usually be reached with the tip of the examining finger.

It is also approximately the level of the mid-rectum. The rectosigmoid will be approximately 15 to 17 cm. from the anal verge (Figure 1).

The inferior mesenteric artery arises nearly two inches proximal to the bifurcation of the aorta. The third portion of the duodenum may course across its origin. After high ligation of this vessel, the blood supply to the colon is carried through the marginal artery (Figure 1). Early and high ligation of the venous return from the rectosigmoid and rectum should be practiced to control venous metastasis incident to operative manipulation of the local neoplasm. This vessel can be found retroperitoneally just underneath the fourth portion of the duodenum, to the left of the spinal column (Figure 1).

HIGH LIGATION OF THE INFERIOR MESENTERIC ARTERY

It has been observed in the experimental animal[2] and also in the human that in most cases the inferior mesenteric artery can be ligated at its origin without subsequent necrosis of the large bowel.[3] In obese individuals, the origin of the vessel may be difficult to find. In some instances where it is felt that high ligation would not be tolerated, it is permissible to begin the soft-tissue dissection at the origin of the vessel and skeletonize it to the level of ligation. Some surgeons have recommended extending lymph-node dissection to include a high ligation and a preaortic and precaval lymph-node dissection from the duodenum to the sacral promontory.

RECURRENCE

Local recurrence following anterior resection has been reported to be as high as 16 per cent, with 10 per cent recurring at the line of anastomosis. This problem of recurrence has been studied from the standpoint of contiguous spread, incomplete removal of the tumor, local venous spread and the development of a new lesion. Incomplete removal occurs infrequently because mucosal spread in the lumen of the bowel is very limited.[4] Local venous spread probably plays an important part in the recurrence of tumors. It has been demonstrated that there may be gross tumor in the veins surrounding the lesion.[5] The colon is notorious for developing new lesions, and this probably accounts for some of the apparent local recurrences in the line of anastomosis.

Many local recurrences in the line of anastomosis have been attributed to intraluminal spread.[6] A number of investigators, particularly Cole, have demonstrated the presence of intraluminal exfoliated tumor cells. These cells are primarily concentrated distal to the lesion. Some of them may be directly implanted into the anastomotic site at the time of operation, as well as being implanted along needle suture tracts. The cells find a suitable pabulum for growth in the raw tissues at the site of anastomosis. It is thought that 70 per cent of anastomotic recurrences may be attributed to intraluminal spread.[7] To obviate this mode of spread, several precautions may be taken. The lesion may be isolated between ligatures early in the operative procedure, and cancericidal drugs may be used to irrigate the distal segment prior to the beginning of anastomosis (Figure 2).

It has been observed that there may be an element of extraluminal spread. Cancer cells may be exfoliated into the peritoneal cavity from the surface of the bowel overlying the tumor. It is recommended that a barrier be applied to the serosa to prevent this type of spread.[8]

It has been demonstrated satisfactorily that the veins near the lesion may contain gross cancer, which can be found on careful dissection of the veins. Cutting through veins in the region of the lesion may lead to implantation of the raw surfaces, with subsequent recurrence. It is conceivable that the use of a local cancericidal agent could inactivate or destroy these exfoliated cells or clumps of cells.

DISTANT SPREAD

Circulating tumor cells in the peripheral blood have been demonstrated in patients with a varie-

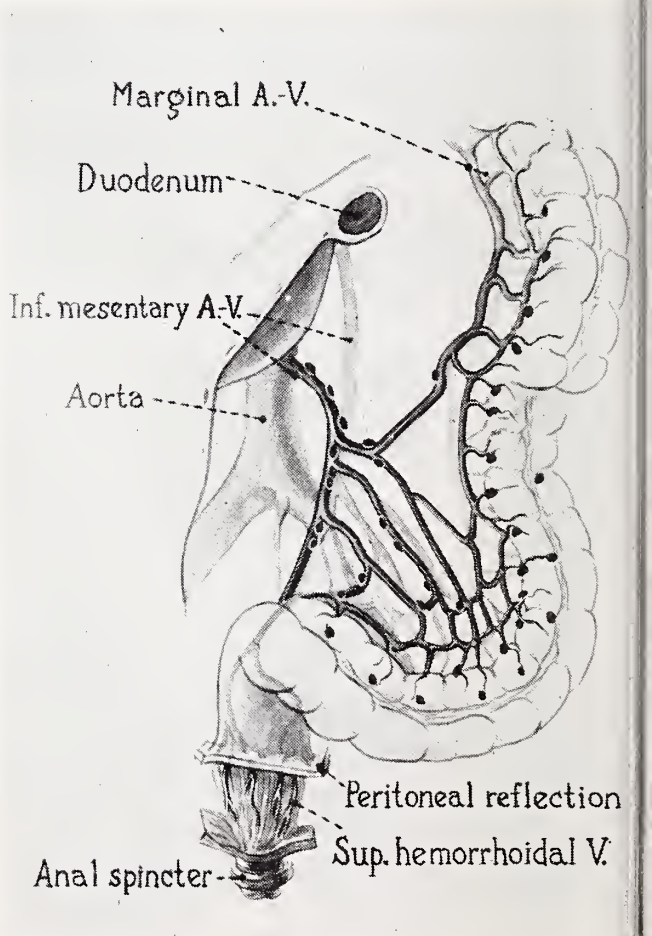

Figure 1. Anatomical orientation.

ty of neoplastic lesions. The farther advanced the lesion, the higher the concentration of tumor cells. In early lesions, tumor cells are seldom found in the peripheral blood.[9] Vein invasion, either microscopic or gross, appears in some 40 to 60 per cent of cases of carcinoma of the rectosigmoid and rectum. This represents a reservoir of tumor cells. It has been noted that a "cell shower" at the time of surgery does occur coincidentally with manipulation of the tumor. The tumor cells in the peripheral circulation apparently remain there for approximately 48 hours before they are fixed in the tissues.[7] To prevent the cell shower at the time of operation, early ligation of the venous return from the area is advocated. It seems logical to try to find a chemotherapeutic agent that could be used to destroy the circulating and unfixed tumor cells following operative removal of the primary.

CHOICE OF OPERATION

Several criteria should be fulfilled when one selects an operation for a patient who has carcinoma of the rectosigmoid or rectum. First of all, he should select a procedure that gives the patient the greatest chance of cure. This may sound axiomatic, but it is felt by many that in recent years there have been some compromises with this principle.[10] The operation should be planned so as adequately to excise the tumor and encompass the predictable lymphatic and venous spread that is capable of resection. The primary tumor may be adherent to contiguous structures so as to necessitate extending the incontinuity resection to include adjacent structures. Occasionally it is justifiable to extirpate a single metastasis from the liver, either at the primary operation or as a secondary procedure.

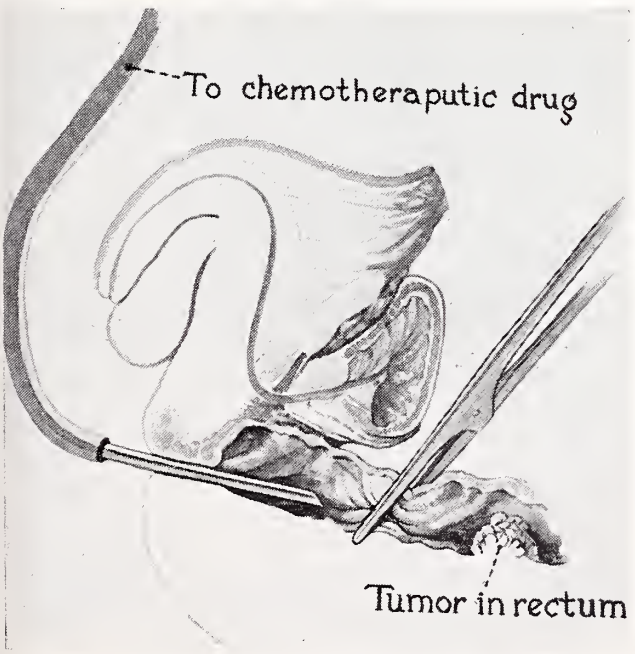

Figure 2. Perfusion of rectal segment.

After all of the above criteria have been satisfied, one can choose a procedure that is least mutilating to structure and function.

The surgical treatment of carcinoma of the rectosigmoid and rectum could be simplified if we were willing to subscribe to the combined abdominoperineal resection as the treatment of choice for lesions in both these anatomic areas. However, other operative technics do have merit. Some of the operative procedures seem to be indigenous to certain geographic locations where the results are uniformly good.

The operative procedures can be divided roughly into two categories: (1) the non-sphincter-saving operation typified by the combined abdominoperineal resection popularized by Miles, and (2) the sphincter-saving operations, which can further be subdivided into anterior resection and abdominoperineal procedures.[11]

The cure rates for carcinomas of the rectosigmoid and rectum decrease as they approach the anal canal. The cure rates as reported are somewhat similar, regardless of whether a sphincter-saving or a non-sphincter-saving procedure is used. It does appear, however, that in some of the series of cases a definite element of selection has been present in choosing patients for sphincter-saving operations, and that most surgeons save the combined abdominoperineal resection for the "big" tumors.

In order to insure "consistent" continence of feces in patients in whom a sphincter-saving operation is contemplated, the anal canal should be left intact (since that is the "sensing" area that helps one distinguish between feces and flatus), and the lower portion of the rectum should be left undisturbed to insure some degree of reservoir function.

Generally speaking, most lesions above the peritoneal reflexion can be adequately excised by anterior resection. This type of operation is certainly recommended for lesions of the rectosigmoid, which are at about the 15 cm. level. An anterior resection with primary anastomosis can be done in this region, and the surgical principles of high ligation of the arterial supply, removal of predictable lymphatic spread and adequate removal of the primary lesion can certainly be accomplished.

It is difficult to appraise the distances involved in some of the anterior resections, especially when such terms as "low" anterior resections and "low-low" anterior resections are employed. Controversy arises over whether the operative approach is anterior or perineal, and it seems that surgical principles are being violated.

A lesion that is palpable from below is one that is at or below the peritoneal reflexion. For these lesions readily accessible to the palpating finger, an extended abdiminoperineal resection (radical) is advocated. This should include wide excision of peritoneum, high ligation of the inferior mesenteric artery, vein dissection along

the lateral pelvic wall, generous removal of skin and ischiorectal fat, and high detachment of the levator muscles.

The highlights of the surgical technic are as follows: (1) early ligation of venous return from the area of the tumor; (2) isolation, when possible, of the tumor between ligatures; (3) high ligation of the major vascular supply to the area to insure adequate removal of lymphatics and nodal lymph tissue; (4) serosal isolation of the tumor, where possible, to prevent intraperitoneal exfoliation; (5) resection of a 2 in. margin distal to the tumor; (6) peritoneal toilet with a cancericidal agent to destroy tumor cells that may have been exfoliated into the operative field; and (7) the use of an effective and suitable intravenous chemotherapeutic drug.

EXTENDED OPERATIONS

An extended operation may include all the pelvic viscera. Although three systems may be involved in this resection, only one requires special reconstruction. That system is the urinary drainage tract. A number of methods have been devised for managing this problem, but the one that has stood the test of time is the ileal conduit.[12]

In forming the ileal conduit, a 6-8 in. length of ileum near its terminal part is isolated with an intact vascular supply. Intestinal continuity is reconstructed, and the segment of ileum that has been isolated is closed at its proximal end. The ureters are then implanted into the proximal portion of the ileal conduit. Vascularity to this area is ordinarily no problem, if the blood vessels have been carefully preserved. A two-layer uretero-intestinal anastomosis without tunneling is usually utilized. One of the essential elements is mucosa-to-mucosa apposition to insure early healing with minimal scar (Figure 3).

There are fewer complications with this type of drainage than with other types—e.g., sigmoid implantation and wet colostomy, cutaneous ureterostomy or transplantation to an isolated reservoir of cecum.

LIMITED RESECTION

A certain number of poor-risk patients with carcinoma of the rectum present tumors which may not be large but can be invasive. The ordinary operation of choice in such a lesion would be the abdominoperineal resection. Operations for preservation of the sphincters are probably equally traumatic to the poor-risk patient. Local treatment of the lesion may be all that is justified. Almost all large series of patients who have been treated for carcinoma of the rectum include a fair proportion of these individuals who have been treated by local destruction. There are several methods available for handling these small lesions: (1) local excision; (2) fulguration; (3) radiation; or (4) a combination of these various measures.

In most series of cases there is a reported 50 per cent five-year survival in this group of patients who have been treated by endo-anal excision, fulguration or radiation. This type of treatment may be satisfactory for carcinoma *in situ* and noninvasive carcinoma in polyps.[13]

INFLAMMATORY LESIONS

Because of the nature of the environment of tumors of the large bowel, they are infected. The inflammatory reaction may spread into adjacent tissues by penetration or perforation. Because of the apparent hopelessness of the situation, a proximal defunctionalizing colostomy is done. Occasionally we are pleasantly surprised to find that the lesion has subsequently so diminished in size that it can be considered resectable.[14] The use of the defunctionalizing colostomy has increased the resectability rate, and at times we find five-year survivors among this group.

A study of the pathological material obtained at the time of pelvic viscerectomy has shown that the adjacent organs have been involved by the

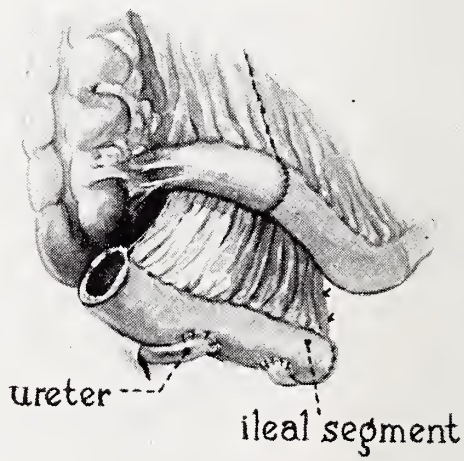

Figure 3. Ileal conduit.

tumor in only about 40 per cent of the cases. The inference is that those cases considered to have bladder, vaginal or uterine involvement are often manifesting abnormalities due to an adjacent inflammatory reaction. By employing proximal defunctionalizing colostomy in those cases where bulky masses are present, we may be able to decrease the number of ultraradical procedures in the pelvis.

PALLIATIVE OPERATIONS

As previously stated, patients with untreated carcinoma of the colon and rectum survive approximately 17 months. Colostomy or bypass operations do not affect this survival rate. Resection of the lesion does increase the survival rate appreciably.

Because of this increased survival with resection, we should try to resect the primary lesion even in the face of distant spread. This may necessitate abdominoperineal resection or even excision of contiguous organs. We should give particular consideration to sphincter-saving procedures when doing palliative operations. It is permissible to violate some of the surgical principles previously elucidated, when one is performing a palliative type of operation.

Operations for palliation in this disease not only increase life expectancy but makes the remainder of the patient's existence more pleasant. By excising the local lesion, we prevent the spread of neoplasm to nerve fibers, with its attendant pain, and we prevent the constant oozing of plasma and blood from the ulcerating primary lesion, thereby slowing the process of malnutrition. Excising the primary lesion may actually slow the rate of metastasis to various organs.

NEW GROWTHS

The mucosa of the colon and rectum is noted for its growth potential. It is not altogether uncommon to note synchronous carcinomas of the colon at the time of operation. Metachronous or new primaries are frequently encountered at the time of operative reexploration of the colon, or of radiologic reexamination. It has been estimated that approximately 30 per cent of the colons resected for carcinoma contain polyps. Many physicians consider the colonic polyp a precancerous lesion.

Extended ablation of colonic segments associated with removal of primary carcinomas has in many instances removed the primary polyp-bearing area, and the operator will occasionally remove a synchronous carcinoma with this extended colonic removal.

COLOSTOMY

Proximal colostomy, of a defunctionalizing type, may be indicated in the poor-risk patient with a high-grade obstruction. It is not, however, done as a routine prior to or at the time of anterior resections with primary anastomosis. Proximal complementary decompression colostomy may be indicated where there is some question of the integrity of the anastomosis.

DISCUSSION

There is a segment of the lower part of the gastrointestinal tract that harbors malignant neoplasms the surgical management of which is quite controversial. The controversy can be resolved into three parts for the sake of discussion. The segment about which there is most disagreement is the lower rectum and mid-rectum. One group contends that the lesions in this area can best be handled by combined abdominoperineal excision of the rectum. The second group advises removal of these lesions via the abdominal route, following an anterior resection, and would classify the procedure as a "low" or "low-low" resection. The third group would use a combined-approach-sphincter-saving operation, with a reconstruction from the perineal aspect. The latter two procedures seem to violate certain surgical principles that were set forth earlier in this paper.

Second-look operations were first popularized for the follow-up in patients with carcinoma of the stomach.[15] It has since been found that the best salvage rate from second-look procedures is in carcinoma of the rectum and colon. Second-look operations are still somewhat in an experimental phase and should be done in controlled series. Fortuitous circumstances often allow second-look procedures that may be informative.

Mention was made earlier that there is a rather high recurrence rate for carcinoma of the colon and rectum. The prognosis following the development of a recurrence is not good, but re-resection should be undertaken in those cases wherever possible.

The advantages of using cancericidal agents in carcinoma of the colon and rectum have not been clearly established. The ideal agent is not available at the present time. Emphasis, recently, has been placed upon local arterial infusion of tumors. In low-lying rectal lesions where it is felt that there has been an inadequate excision, consideration should be given to intubating the hypogastric arteries with small catheters and bringing them to the body surface for the purpose of subsequent infusion of a cancericidal agent into the area, after a method used for carcinoma of the cervix.[16]

In anterior resections where reconstruction of the intestine is to be done, several methods of anastomosis are available. Most surgeons use the conventional, open, two-layer closure consisting of an inner layer of continuous catgut and an outer layer of interrupted nonabsorbable suture.

In recent years, we have employed a complex suture that has proved useful. This suture was devised by Gambee and has essentially deep and superficial components, but by the method of its placement it insures mucosa-to-mucosa as well as serosa-to-serosa apposition (Figure 4).

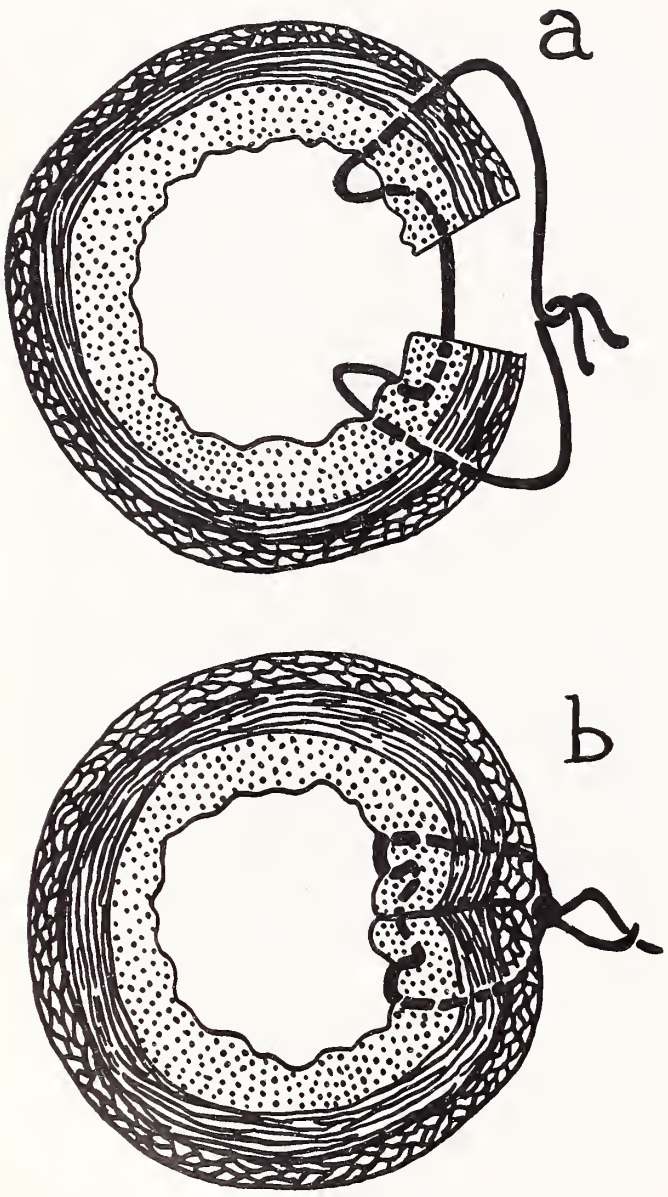

Figure 4. Gambee suture.

Antibiotics in solution are sometimes used to bathe the suture line during the anastomosis. A portion of this solution may be instilled into the proximal and distal segments from the area of anastomosis just before the latter is completed.

SUMMARY

1. Surgical treatment of carcinoma of the rectosigmoid colon and rectum seems to have reached its high point, and additional technical changes probably will not contribute much toward increasing life expectancy.

2. Attention should be directed toward the prevention of recurrences.

3. Radical but reasonable treatment of these lesions is indicated, and should be pursued hopefully until distant spread has been proved.

4. Anterior resection is advocated for lesions at and above the rectosigmoid junction.

5. Combined abdominoperineal excision is the treatment of choice for lesions readily accessible to the examining finger.

6. Local ablation of accessible lesions is reserved for poor-risk patients.

7. Palliative resections are justified, for they prolong life and provide comfort.

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The Bierring Fund is to be a part of the IMS Educational Fund, Inc., from which loans are made to help medical students complete their education.

Additional contributions, of whatever size, will be welcomed.

Pancreatic Surgery

WILLIAM J. GILLESBY, M.D.

HINES, ILLINOIS

In the mid 30's the disease of pancreatitis was considered to be a disease of inebriates. The acute form was seen in various charitable institutions and among the lower strata of society. In recent years, however, we have been seeing these acute and chronic forms of pancreatitis resulting from other conditions. Chronic relapsing pancreatitis following long bouts of biliary tract disease is being recognized more frequently, and it is somewhat disconcerting to all of us physicians that the pancreatitis following poorly done peptic ulcer surgery is becoming more common.

The pancreas produces at least four secretions: insulin in the islet tissue, trypsinogen, amylase and lipase and perhaps several others that we don't know about. Literature is becoming loaded with reports, each one mentioning the discovery of a new enzyme. The extrinsic secretion of the pancreas consists of about 1,000 cc. of fluid daily. This helps to digest protein, carbohydrate and fat. Trypsinogen must be activated by enterokinase to become trypsin, but the others require no activation as far as we know.

Pancreatitis is the result of some insult to the pancreas, with partial or complete blockage of the duct by stone, edema, spasm or chemical insult. Alcohol seems to be the most frequent of these latter insults. A large number of our patients who developed acute pancreatitis, drink poor liquor. We analyzed some of the whiskies from Skid Row in Chicago and found out that some of them ran as high as 3-5 per cent methyl alcohol. The beverages were tainted by denatured alcohol or anti-freeze or something else so that the price could be cut. We know that methyl alcohol produces acute pancreatitis. It also produces acute optic atrophy. If methyl alcohol is mixed with a large amount of ethyl alcohol, the effect of the methyl alcohol is retarded, it takes effect more slowly, and its effect on the pancreas is probably less lethal. We believe that pancreatitis in the alcoholic group is more common among individuals who drink cheap liquors. This last is intended to make you feel a little better when you have your martini each evening.

ACUTE PANCREATITIS

The signs of acute pancreatitis are elevated amylase, lipase and prothrombin, or the so-called heparin test, but they are so variable that I don't know what to tell you about evaluating their results. Harkins and others said, years ago, that if the amylase level is over 500, the patient has acute pancreatitis. We have found in recent years how wrong they were. For instance, in mesenteric thrombosis or a vascular accident to the small bowel, the amylase may be up over 1,000, and at postmortem the patient may be found to have had a vascular situation and to have had nothing wrong with his pancreas at all.

We are, I think, agreed that the surgical treatment of acute pancreatitis has twice as high a mortality rate as does medical treatment. The problem then is to know when to operate and when to stay out. An alcoholic history is of some help. Fever and pain—especially *relentless* left-back pain—seem to be about as common as any others of the signs. The pain is severe. I've noticed one thing which is not 100 per cent reliable. If you sit on the patient's bed, he protests if he has acute pancreatitis, for he does not wish to be jiggled. The test isn't infallible, but if you sit on the patient's bed and he rather wishes you'd get off, you'd better suspect that he has acute pancreatitis. Shock is pronounced.

One of the things that all of us have done—and I think it is rather important—is to aspirate the abdomen. I don't advise you to attempt it if you never have done it before. Rather, perhaps, you should try it first on a cadaver—a postmortem case—so as to get the feel of how to go in and get some abdominal fluid. Aspiration may not be of great help, however, for in the bloody fluid of mesenteric thrombosis, the amylase may be just as high as in acute hemorrhagic pancreatitis.

The treatment of acute pancreatitis is usually epidural block. Splanchnic block is too hard to do, and I think epidural blocks work better. You had better call the anesthesiologist for it, but if you don't have one whom you can call, splanchnic blocks will work. Nitroglycerine under the tongue, or amyl-nitrite, may help relieve the spasm, and you must start nasogastric suction. Calcium gluconate is given, frequently, regularly and almost indiscriminately, 10 cc. every four hours. Just keep on giving it; you can't seem to give too much. Procaine had better be given by the nasogastric tube, and you can use procaine intravenously. I have found it quite helpful, and it permits one to cut down on the amount of narcotic that one uses. Streptomycin is the antibiotic of choice because the pancreatic fluid secretes it better than any of the other antibiotics.

Belladonna and its derivatives should *not* be

Dr. Gillesby is assistant chief of the Surgical Service at the V. A. Hospital in Hines, Illinois. This presentation was given in Davenport, at a meeting of the Scott County Medical Society, March 7, 1961.

used. Every detail man in the business will tell you that they cut down the secretion. They cut down the volume, but they do not cut down the solid amount, and all that they do is to change a rather thin secretion into a thick syrup. Thus, it can't flow freely, and in our institution we have precipitated three cases of acute pancreatitis where there was a pancreatic fistula. We dried up the fistula, but we plugged up all of the ducts, and since the pancreas could not empty well, the patients developed acute pancreatitis. Belladonna and its derivatives increase the patient's discomfort; he begins to realize that he is really sick, for his mouth is extremely dry; and rather than having done anything worthwhile for him, the physician has made the disease worse.

If you open the abdomen, what do you do? The first thing I do when I open the abdomen and discover acute pancreatitis is to turn to the patient and say "Oops, sorry!" But he is asleep, so I am in no danger from a medico-legal standpoint. Then I do what all teachers do; I turn to the resident and scold him. After getting through with those two little rituals, I try to drain the gallbladder or the common duct, do a Foley tube gastrostomy and then drain liberally.

I don't want this patient to have to have a nasogastric tube down irritating his esophagus and pharynx for the week that it is going to take for him either to get well or to die. The individual with a nasogastric tube in place is one of the busiest people in the world. He spends 24 hours a day gulping, trying to get rid of that throat irritation. What happens? There is an effusion of pharyngeal fluid, and an effusion of esophageal fluid, and the amount may be as high as 2,200 cc. You take out the nasogastric tube and test him with the gastrostomy tube, with no nasogastric tube in place, and the average secretion will be around 750 cc. So when you use a nasogastric tube, don't think that it is the most innocuous little instrument that you have. It is a rather nasty thing, and if any of you have ever been sick and have had to live in bed with a nasogastric tube in place, you must have come to the conclusion that there are things worse than death. In addition, the nasogastric tube permits acid gastric juice to come up along the tube between the wall of the tube and the esophagus, and to produce inflammation esophagitis.

We merely make a stab in the stomach and put in a Foley catheter. We use a 24 Foley with a 5 cc. bag. We blow it up, pull out the mucosa, tie it off and do a purse-string as in the Stamm method, bring it out through a stab wound in the side, and suture the stomach to the anterior abdominal wall. It is very comfortable, I can assure you.

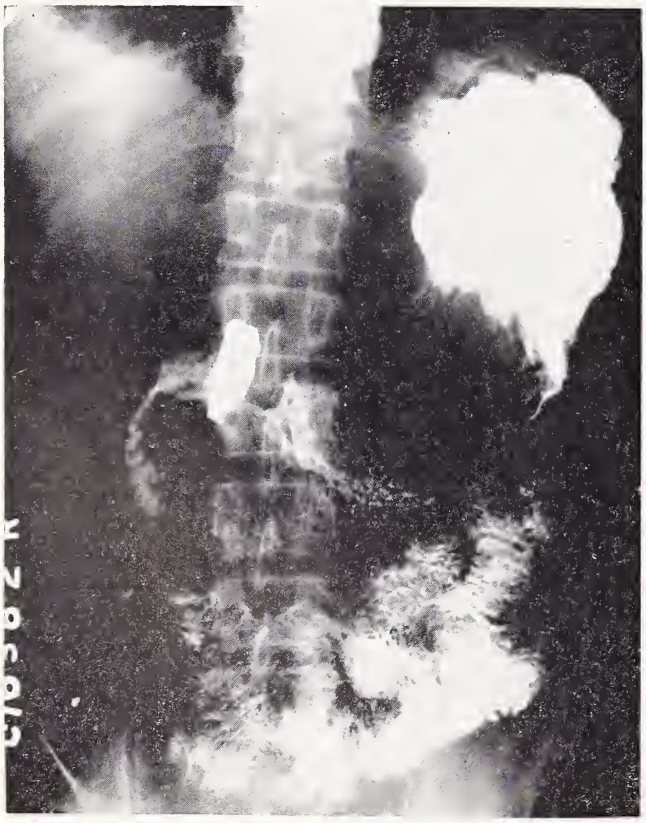


Figure 1. Upper gastrointestinal series shows pressure on the antral portion of the stomach, with flattening of the mucosa. This pressure was from a pseudocyst of the pancreas, posterior to the stomach.

Figure 2. Upper gastrointestinal series shows widened duodenal loop, as well as pressure on the greater curvature of the stomach, pushing the two surfaces together. This was a very large pseudocyst of the pancreas.

ABERRANT PANCREAS

One of the things that will fool us from time to time is an aberrant pancreas. We see it every now and then. I think I have had three this year, and this is only March, so they occur fairly frequently. Why bits of pancreas can be found in these odd places, I have no means of knowing. They may bleed; they may produce obstruction. We found a pancreatic ring, the other day, around the duodenum. We went in thinking that it was carcinoma and found that it was aberrant pancreas. We have even had pancreas occur twice in a Meckel's diverticulum, and it can be found from the esophageal hiatus to the cecum. I don't want to dwell upon these rare conditions, except to say that you should keep thinking of the possibility of them.

COMPLICATIONS OF ACUTE PANCREATITIS

We come to some of the complications of acute pancreatitis. The most common of them, I would say, are chronic relapsing pancreatitis or pancreatic pseudocysts. If you find a mass in the abdomen and at surgery you think it is a pancreatic cyst, you *must* aspirate it. A surgeon, many years ago, found a mass in the abdomen which he thought was a pancreatic cyst so he put in a trochar, attached it to the suction apparatus, and emptied the "pancreatic cyst." There was just one little difficulty. It was an abdominal aneurysm. It emptied well! Thus, I want to stress that whenever you are in doubt, be sure to aspirate. What color are the fluids? They may be black, green or any other color. One can't predict this. They come out in odd fashions.

The old treatment was to marsupialize, and it still has to be done where the wall of the pseudocyst isn't a substantial one. We have one trouble with it because a large amount of electrolytes are lost, and the patients feel tired all of the time. They are so low on potassium and so low on the other electrolytes that they feel exhausted. Thus, we feel that pancreatic pseudocysts should be emptied into the bowel in some fashion.

Here is a pseudocyst pressing onto the stomach (Figure 1). I don't think I need tell you that there is pressure there.

Here is a very beautiful example of a pseudocyst (Figures 2, 3). Here is the cyst pressing from behind—pressing the stomach forward (Figure 4). The lateral is important in all of these. If it presses the stomach backwards, it is a tumor of the liver, not a cyst of the pancreas.

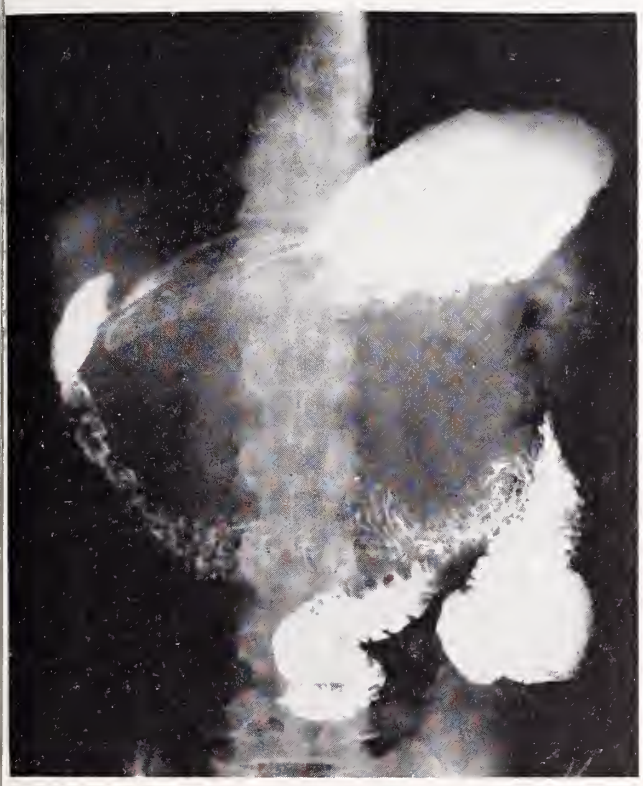


Figure 3. Upper gastrointestinal series showing widened duodenal loop and flattening of the antral portion of the stomach, with distortion of the duodenal cap and the duodenum.

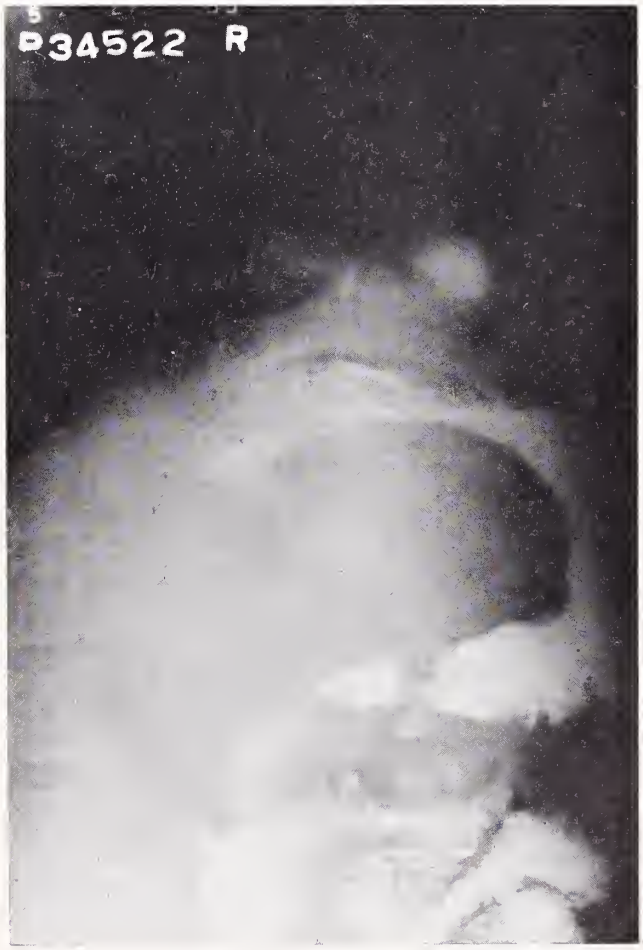


Figure 4. A lateral view of an upper gastrointestinal series, showing the stomach to be distorted by a pseudocyst of the pancreas posterior to the stomach. Note the large size of this pseudocyst.

These cysts will flatten the stomach in various ways, and the x-ray people can tell us more about them than we can discover by clinical symptoms and tests. In this next instance, the stomach was being pressed from behind (Figure 5).

How much will one of these cysts hold? Oh, up to 3,000 cc. They are tremendous. They are usually palpable, but they can be a little bit of a problem. Here we have another one. This one is in the sweep of the duodenum, is protruding between the stomach and the duodenum, and is stretching it (Figure 6). How does this occur? It's a collection of pancreatic juice, out of place. It has wandered from home, and has produced a chemical peritonitis with walling of the cyst.

Why do we call it a "pseudocyst"? Because the wall is its own on only one side. The wall is only what Nature has built up as a defense to keep this fluid in one place. This isn't a cyst wall resembling the walls of other cysts; this is peritoneal reaction, fibrin. The spleen, the stomach, the colon —whatever happens to be near—is the wall of the cyst. If someone says that a pancreatic pseudocyst should be removed, be gentle to him, for he hasn't much sense. It *can't* be removed! It has a wall of its own on only one side!

A pancreaticocystogastrostomy is one procedure for its treatment. A pancreaticocystojejunostomy of the Roux-Y type is probably the best of the group of procedures—just anastomosing the cyst to the jejunum, and then doing a Roux-Y below, trying to get it emptying out as it should. The reason for doing this, rather than marsupializing, is that one attempts to keep the pancreatic juice in the body, rather than having it drain away to the outside.

In chronic relapsing pancreatitis we may have calcification in the pancreas, and this is not calculus. This is calcification. These aren't stones; these are calcium. They are not in the ducts; they are in the tissue itself. By this I mean that the calcification of the pancreas has no similarity to gallstones, renal stones or bladder stones, for these others are precipitations. These calcifications in the pancreas are replacement of tissue. The calcification doesn't at all follow the line of the ducts. These are the replacements of fat necrosis and have become calcified. First, they are calcium soaps and later, calcium salts.

Figure 5. Upper gastrointestinal x-ray showing flattening of the stomach against the anterior abdominal wall due to a tremendous pseudocyst of the pancreas pressing from behind.

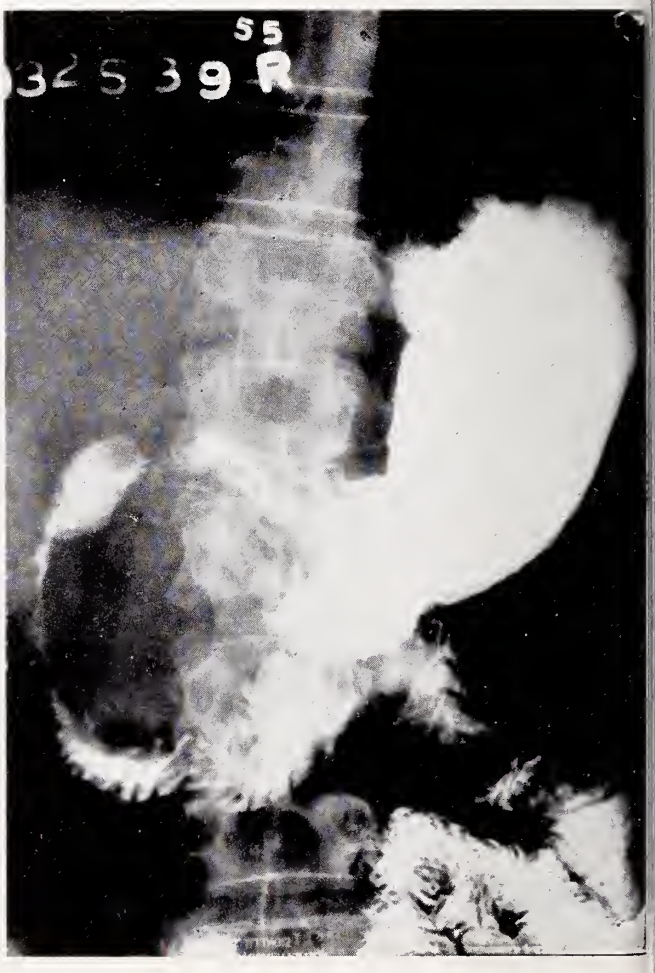


Figure 6. Pseudocyst in the head of the pancreas. This picture shows widened duodenal loop, flattening of the antral portion of the stomach and marked flattening of the duodenal components as well. The distortion is quite marked.

CHRONIC RELAPSING PANCREATITIS

We come to the question of what to do about chronic relapsing pancreatitis, and I won't dwell too long on this because it is like riding a hobby. But we have found out that chronic relapsing pancreatitis is growing more common. There are alternate dilatations and strictures of the pancreatic duct, and we are told that all we have to do is to go in and do a sphincterotomy. In a sphincterotomy one would cut 1-2 cm. at most, but it wouldn't have a thing to do with all the strictured duct. There is a stenosis in the duct up to 8 cm. from the ampulla. We often have a "chain of lakes" effect (Figure 7).

Pancreatograms have been very interesting to us in that they show that the entire pancreas shares in this disease. This isn't just something at the head of the pancreas. This is throughout the entire length of the pancreas, in the ducts and in the tissue. To talk about sphincterotomy is to be deluded, and to think that you can accomplish something by draining a very tiny portion of the ductal system is a hope unfulfilled.

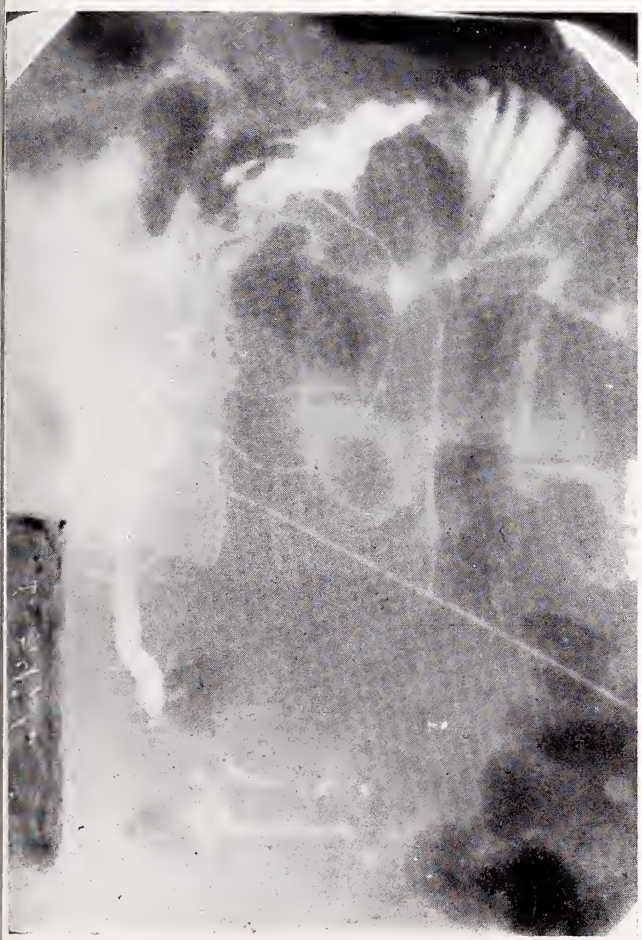


Figure 7. Retrograde pancreatogram following a pancreaticojejunostomy, showing the dilation of the pancreatic duct with alternate stenoses and dilatations—the "chain of lakes" effect. Note that the duct of Wirsung is very long and is stenosed in many areas before it reaches the ampulla of Vater. Sphincterotomy cannot benefit such a condition.

We have designed certain diagrams which represent what the ductal system looks like in chronic relapsing pancreatitis (Figure 8).

In treating chronic relapsing pancreatitis, we slit the pancreas open just like the pages of a book, and lay it open so that it will drain thoroughly.

The pancreas has been laid open, and in three places there are stenotic areas in the duct so

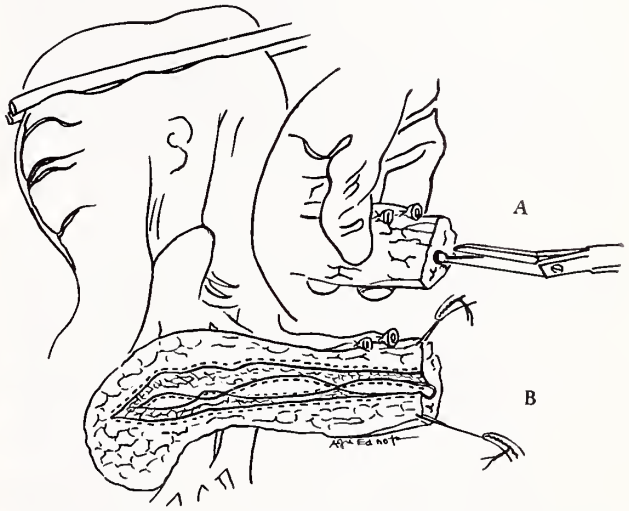


Figure 8. Stomach mobilized and elevated for exposure of the pancreas. A. Pancreatic duct opened with bandage scissors. B. "Chain of lakes" appearance of pancreatic duct.

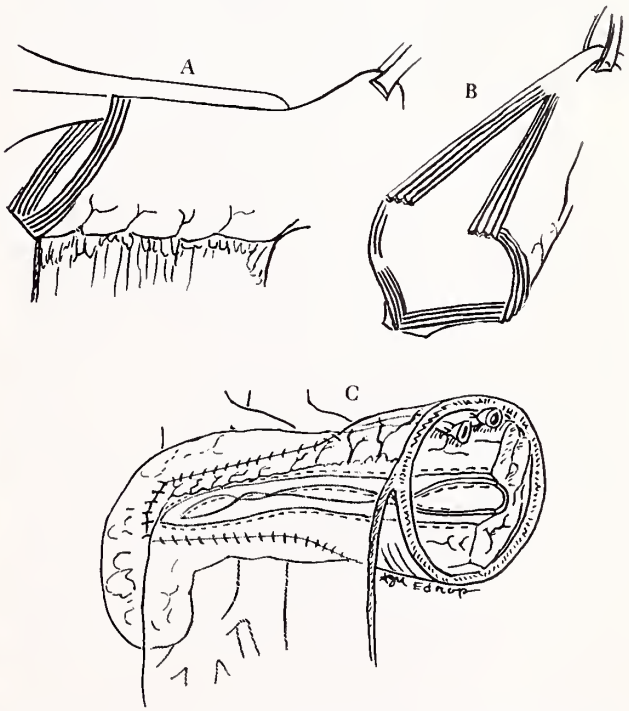


Figure 9. Preparation of jejunum for pancreaticojejunostomy. A. Payr clamp crushing antimesenteric jejunum. B. Division of jejunum in crushed area (3-4 inches). C. Pancreaticojejunostomy—complete coverage of the opened pancreas by opened jejunum.

that the fluid couldn't get from one section to another. As a result, we see that to talk about sphincterotomy is rather ridiculous. You are draining just one tiny little piece of the pancreas and not getting any others.

The pancreas, by the way, is quite fibrotic and scarred. It doesn't bleed as much as you might think. We expose the pancreatic duct as a result of slitting it open with a pair of bandage scissors, and then laying an open piece of jejunum over the pancreas, we suture it in place by a pancreaticojejunostomy (Figure 9). A Payr clamp is used on the antimesenteric border and crushed. We lay this opened jejunum over the open pancreas, suturing it in place, do a Foley gastrostomy, drain and restore the continuity of the bowel by doing a Roux-Y.

ADENOMA OF THE PANCREAS

We come to one other thing that must be kept in mind, and that is adenoma of the pancreas. We have had a fair number of them at Hines. If you recall the Rex Morgan cartoons, of about two years ago, I think it was, there was a character who developed a B adenoma of the pancreas and the series ran for around six weeks. Here is one. This man was committed to an institution because he was "crazy," and somebody had the bright idea of running a blood sugar, and found that it was down to 20. He was given glucose, and he immediately came out of his coma, but then he promptly returned to coma, and this went on and on. So much glucose is given these patients as treatment that they become quite obese and difficult to handle.

CARCINOMA OF THE PANCREAS

We come to one of the things that worry me, and it is carcinoma of the pancreas. We see patients with jaundice, and with a mass in the head of the pancreas, so we go ahead and do a pancreaticoduodenectomy. I think that no pancreaticoduodenectomy should be done without a positive diagnosis—paraffin! Don't trust a frozen section! We have been fooled too often. It is a little bit disturbing to have a pathologist say, "It looks malignant; it is suspicious." That word *suspicious* is annoying me. It is being applied to breast tumors and to carcinomas of the stomach, and it is I who sit on the hot seat. I must do something. I can't be satisfied with the pathologist's calling my specimen "suspicious." But however that may be, I think no major surgery on the pancreas should be done without a positive paraffin-section diagnosis.

What are some of the things that will lead us to think that carcinoma of the tail of the pancreas is present? Migrating thrombophlebitis is one of the commonest. There have been patients in whom both carcinoma of the lung and of the pancreas have worried us, because they had migrating thrombophlebitis. If carcinoma of the head of the pancreas is suspected, I think that we should probably do as much as we can in getting some glands so that the pathologist can give us a diagnosis, and then do a cholecystogastrostomy, if the gallbladder is still present, and start draining the biliary tract. Then, when you do your pancreaticoduodenectomy, split the rest of the pancreas open so wide that you can really cover it up by a segment of bowel, much as I have suggested, and your incidence of fatality will go down. We are beginning to think that we are making some progress in this pancreatic surgery, by literally laying the remaining pancreas open and letting it drain thoroughly. If we try to cover it up—to suture the pancreas—we get into trouble. But if we can cover it with bowel or with stomach—and stomach isn't as good as bowel, but if one must, he can use it—the recovery is easier.

If you have a case of carcinoma of the head of the pancreas with metastases, jaundiced, probably you'd better do a cholecystogastrostomy as your preliminary procedure. Then, three weeks later, do a pancreaticojejunostomy to drain away this fluid, and you will be delighted to find out that the pain is relieved. The patients don't live any longer, but they live comparatively happily for the length of time that they survive.

SUMMARY

I've given you some ideas on surgery of the pancreas. I've talked rather glibly about pancreaticojejunostomy, and I have seen a certain amount of mystification in the faces of some of you. These operations aren't easy. I don't know how to simplify my presentation of this surgery much more than I have been doing. But if you are going to do radical surgery for carcinoma of the head of the pancreas, you're going to wish you hadn't in many, many cases. The results aren't very good. The cures are so infrequent that one's chances are negligible, and those patients who do survive—and some of mine have survived for about three years, now—may wish that you hadn't prolonged their lives. They won't be very happy. With all of their reconstruction, they will be having some troubles.

Chronic relapsing pancreatitis patients are the most grateful. The pseudocysts are grateful. Those who have had adenomas of the pancreas, the hyperinsulinomas—they too are wonderful, although you aren't going to get them completely back to normal. But as for the people who have had the glucose down as low as 20, you may be able to relieve their pancreatic hypersecretion of insulin, but the damage to the brain is permanent and it is rather pitiable. Some of them remain somewhat retarded mentally, and though they are not so bad as they were, they don't come right back and are not completely new people.

Introduction to Medical Genetics, Part II

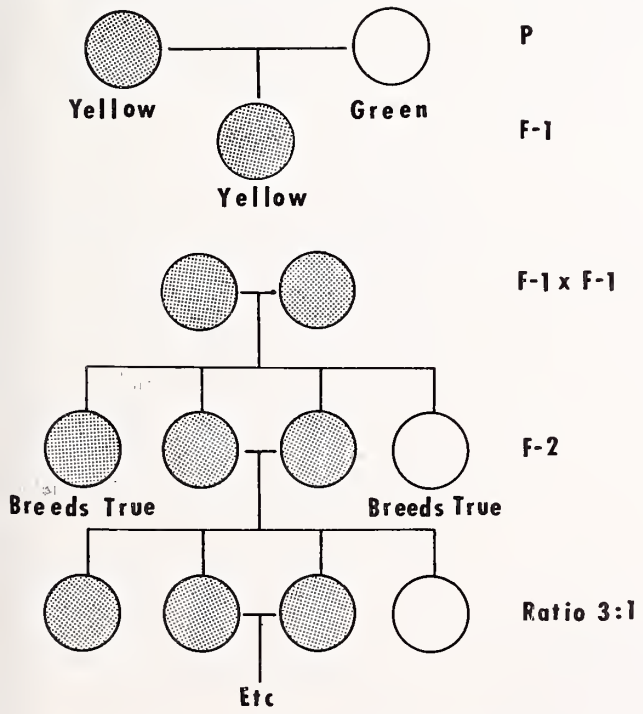
The Cytologic Basis of Heredity

JOHN M. OPITZ, M.D.
MADISON, WISCONSIN

One hundred years ago, Gregor Mendel, a monk and later the abbot at the Monastery of Brünn, in the old Austro-Hungarian Empire, performed a series of experiments that are regarded as the cornerstone of all genetics. Their importance, however, was not recognized, and as a matter of fact the world was unaware that they had been performed until 35 years later, when Tschermak, Correns and DeVries rediscovered them almost simultaneously in 1900. Soon afterwards, histology and cytology had progressed sufficiently to permit their being synthesized successfully with genetics to form that branch of biology known as cytogenetics.

In one of his experiments, Mendel crossed two lines of pure-breeding peas by artificial cross-pollination. These were a variety with yellow seeds and another with green seeds (P = parent generation). All the peas resulting from this cross were yellow (F-1 generation). These results could allow the conclusion that the factor responsible for the transmission of green color had either disappeared or had been masked by a stronger yellow factor. Taking these yellow F-1 offspring and crossing them by permitting self-pollination, he obtained a total of 8,023 peas, of which 6,022 were yellow and 2,001 were green—a 75 per cent to 25 per cent or 3:1 ratio in the F-2 generation. This experiment convinced Mendel that the green color factor had indeed been masked in the F-1 peas by a stronger yellow factor, and that the two color factors, instead of mixing, had associated freely in F-1 and

Part I of this presentation appeared in the July, 1961, issue of the JOURNAL. The editors regret having made a mistake, in a footnote accompanying the first installment, as regards Dr. Opitz' post at the College of Medicine of the University of Wisconsin. He is a resident in pediatrics there.



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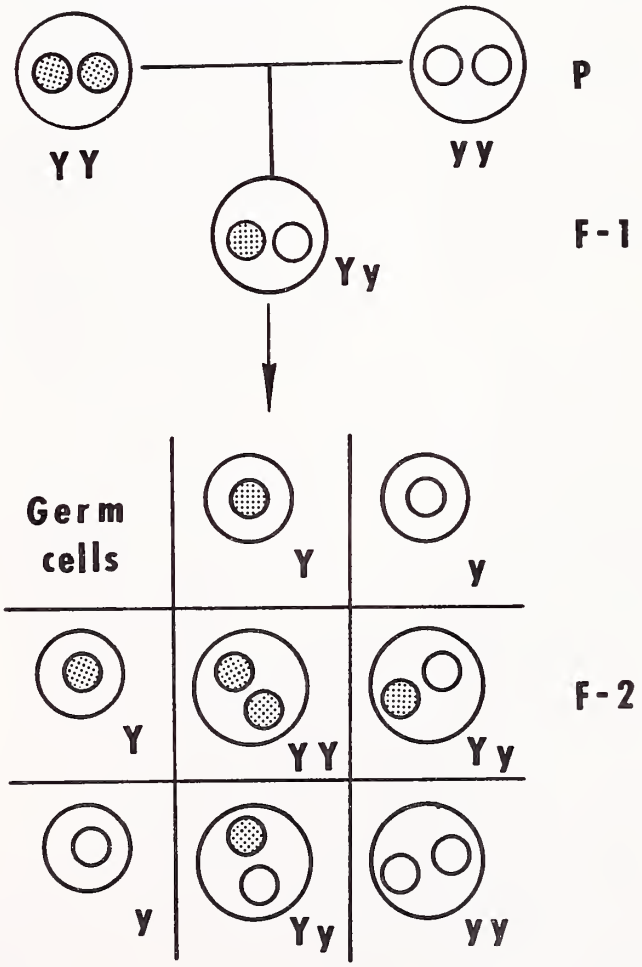

1B

Figure 1. Derivation and recapitulation of basic mendelian principles. Initial cross between peas with yellow and with green seeds. The test cross would involve F-1 (Yy) and the recessive parent (yy), and would yield a 1:1 ratio of offspring (Yy and yy).

had been able to separate independently in F-2 again. In a later cross, he tested the F-2 peas and found the green ones to be a pure strain. However, only one-third of the yellow ones bred true, and two-thirds of them yielded again a 3:1 ratio of yellow and green offspring. The factors controlling the color of peas were thought by Mendel to be transmitted through germ cells. Figure 1A recapitulates these experiments, and in Figure 1B they are explained logically on the assumption of two (i.e., a pair of) genes.

The *test cross* that is always performed to prove Mendelism involves the F-1 individual and the recessive parent. A 1:1 ratio of offspring should always be obtained.

These experiments established the two important mendelian principles of *independent assortment of non-alleles* and *free segregation of alleles,* as well as the following concepts and definitions:

Dominance is the term Mendel applied to the ability of one factor (gene) to determine the color of a pea in spite of its association with a second factor which produces a different character in pure-bred strains. The inability of the green factor to express itself he called *recessiveness* or *recessivity.* Variations of this concept were discussed in the first part of this presentation as incomplete or partial dominance, or recessivity.

The *phenotype* is the appearance of the organism as contrasted with its genetic constitution or *genotype.* These are not always synonymous, as can be seen in the two types of yellow peas in the F-2 generation (*YY* and *Yy*).

Homozygous and *heterozygous* refer to the genetic purity of heterogeneity of an organism for a certain trait. Thus, the pure-breeding F-2 plants, producing only yellow and green peas, are homozygous for their respective color factors, but the type which produces a 3:1 ratio of offspring (in the same manner as the F-1 hybrid) is heterozygous in that it contains both yellow and green factors (Yy).

Diploidy is the genetic state of the somatic and gonadal cells in the sense that these contain a double complement of genetic factors (2n), one derived from the father and the other from the mother. The gametes (sperm and ova), however, must contain only one set of genetic factors (n). This is the *haploid* state (i.e., *Y* or *y,* as in Figure 1B).

The experiment using only one pair of characters is known as the *monohybrid cross.* The same reasoning applies to a *dihybrid cross* using two traits which associate and segregate freely (*A* and *B* dominant over *a* and *b*). If the parents are *AABB* and *aabb,* they produce only two types of gametes: *AB* and *ab,* respectively. All of the F-1 offspring are *AaBb,* displaying the phenotype of the dominant parent.

Assuming free segregation of all traits, four types of germ cells are possible from F-1: *AB, aB, Ab* and *ab.* If a cross is performed between two of these F-1 hybrids, a 9:3:3:1 ratio of offspring is obtained in F-2; i.e., there are nine individuals having both dominant characters *A* and *B* in various combinations (*AABB,* 2 *AABb,* 2 *AaBB* and 4 *AaBb*), there are three of each type displaying only one of the dominant traits each (type *B* only: *aaBB* and 2 *aaBb,* and type *A* only: *AAbb* and 2 *Aabb*), and finally there is only one double recessive (*aabb*) in the total of 16. Crosses using three, four or more characteristics may be performed on the same basis, and have been used extensively to create new and to modify old varieties of plants and animals.

ALLELISM

Whenever two traits are found which obey the segregation law, they are called *alleles.* Alleles, then, are pairs of genes situated in identical positions on homologous members of pairs of chromosomes, one of each pair being derived from the mother and the other from the father. In most instances, the two alleles "behave" normally, i.e., they carry out their usual genetic functions without calling attention to themselves. However, if one or both members of a pair are mutations, their abnormal action becomes obvious. In this way, we have obtained much insight into abnormal and normal function and structure as well. The association may then consist of a pair of normal genes, of a normal one and a mutation, or of two abnormal genes.

In many instances, several alleles may exist for a single locus, as for example in the blood groups in man (genes A, B, and O; allelic pairs: AO, AA, BO, BB, AB and OO), and coat colors in the rabbit. In those cases where the normal allele is dominant over a recessive mutation, a silent carrier state exists and may be maintained over several generations until a homozygous state results from the mating of two heterozygous carriers. Only then are glimpses obtained of the prevalence of

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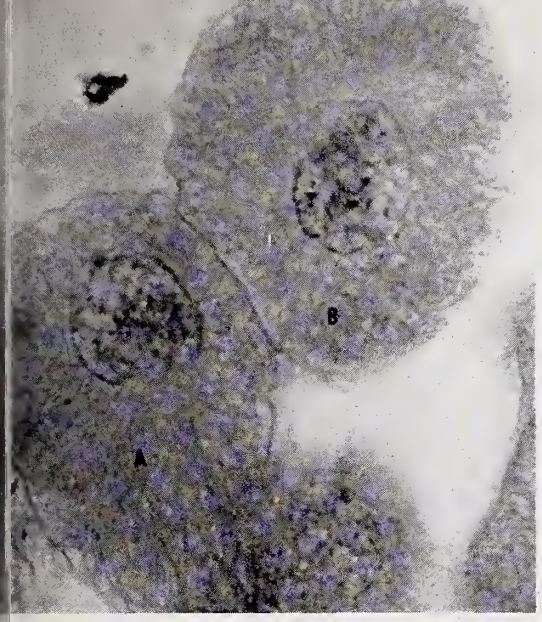
Figures 2 through 9 show the stages of mitosis in the whitefish blastula. Figure 2. Interphase and early prophase. Cell A shows the characteristics of the resting cell, with finely dispersed granular chromatin. In cell B, chromatin is more clumped, and there are two asters visible at almost opposite poles of a kidney-shaped nucleus.

Figures 3 and 4. Prophase. The chromatin condenses into chromosomes, the spindle forms, and the nuclear membrane begins to disappear. The darker spots on the chromonemic-chromosomal fibers are the chromomeres.

Figure 5. Metaphase. Chromosomes are aligned equatorially across the spindle. Spindle fibers attach to each centromere so that the arms radiate centrifugally in a V- or J-shaped fashion.

Figures 6, 7 and 8. Successive stages of anaphase. In Figure 6 the centromere holding the chromatids together has divided, and it can be seen how the daughter chromosomes separate and move to opposite poles, with the tips of the arms still in contact. In Figures 7 and 8, the chromosomes are approaching their destinations, and the cell is beginning the process of cytokinesis.

Figure 9. Telophase. This photo shows especially beautifully the resolution of the chromosomes into a whorl of chromonemata, the hourglass shape of the remnant spindle and the reformed cell membrane separating the two daughter cells.



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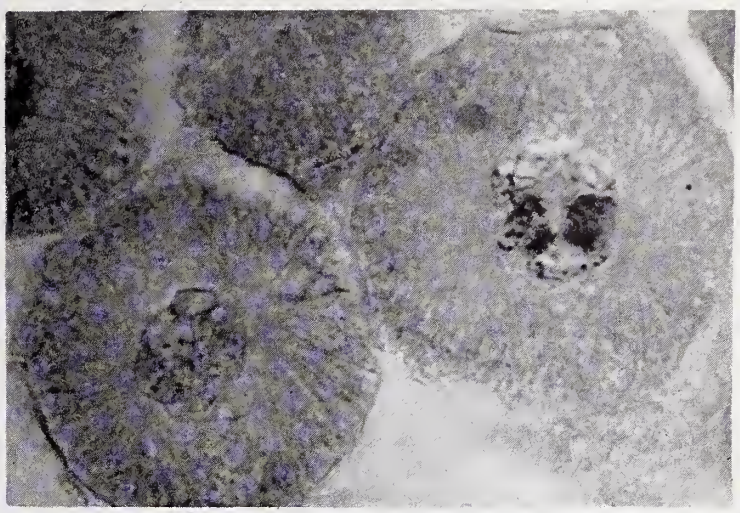

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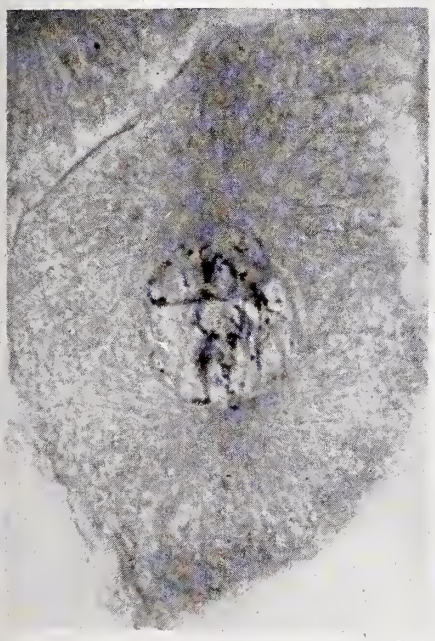

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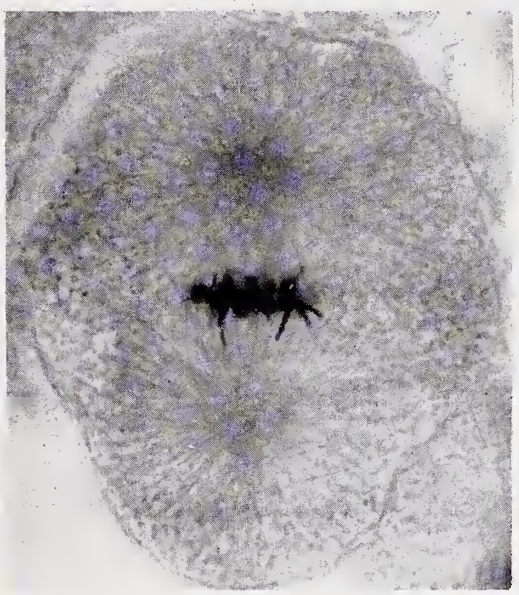

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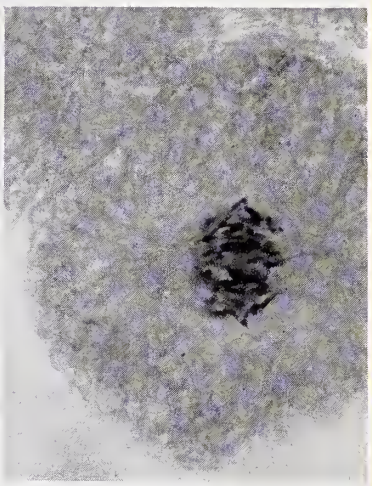

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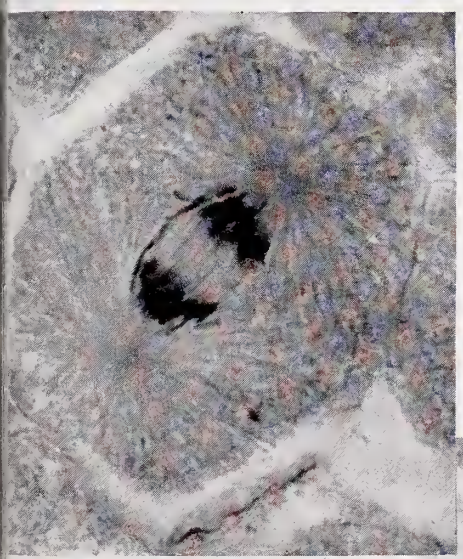

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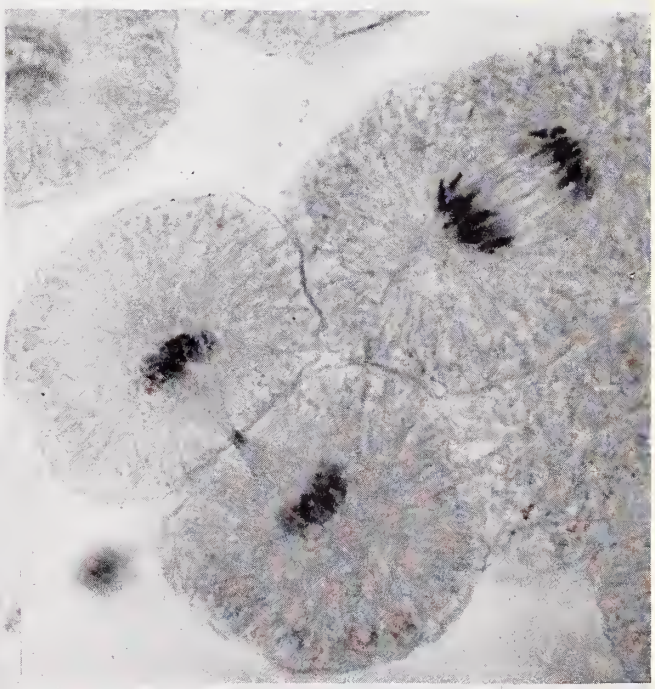

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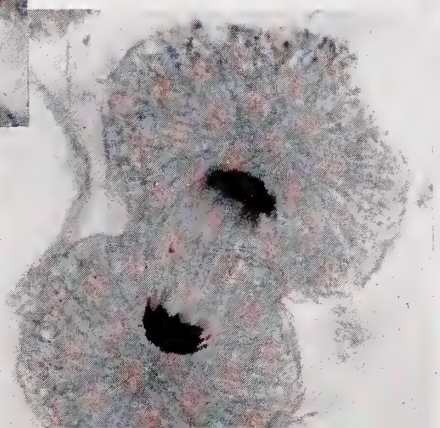


recessive mutations hiding under the cloak of anonymity of their normal alleles.

LINKAGE

The early science of genetics had an atomistic basis, presupposing the independent existence, assortability and segregability of all genes. In 1906, however, Bateson and Punnett found an exception in their work with sweet peas. Studying two factors influencing pollen shape and color of blossoms by breeding homozygous dominant with homozygous recessive plants (*AABB* and *aabb*), they attempted to reproduce a dihybrid cross with a 9:3:3:1 ratio of offspring from the F-1 generation *ABab* (assuming the production of germ cells *AB*, *Ab*, *aB* and *ab*). This did not work. Instead, they produced only two types of plants in a 3:1 ratio (AABB, AaBb, AaBb and aabb). Thus each pair of characteristics did not assort independently, but tended to enter the same germ cells and to be transmitted together if derived from the same parent. If the same pair of alleles came from different parents (*AAbb* and *aaBB*), they tended to enter gametes separately and to stay apart. The first phenomenon they called "coupling," and the latter they called "repulsion." Both were later shown to be two aspects of a single phenomenon—*linkage*. This means simply that two factors are inherited "together" in disobedience to the mendelian laws of independent assortment and free segregation, in consequence of their residence in variable proximity on the same chromosome.

By the time the first example of linkage was described, the process of mitosis was well known, and the similarity between chromosomal mechanisms and the movement of the "color factors" or genes of Mendel had caused cytologists to place the genes on the chromosomes (Sutton and Boveri). It was obvious by then that there were far more genes than chromosomes, and the work of T. H. Morgan and his many brilliant collaborators and students on the fruit fly, Drosophila melanogaster, established the fact that sets of genes are usually inherited in numbers of groups corresponding to the numbers of chromosomes—the so-called "linkage groups." Thus, for D. melanogaster, which has four pairs of chromosomes, he determined four linkage groups, and placed the genes on the chromosomes in a linear order determined by the phenomenon of crossing-over (see below). He proved that the tendency of linked genes to stay together was due to their residence in the same chromosome, and that the degree or strength of linkage depends on the distance between linked genes on the chromosome (Sinnott).

MITOSIS

Mitosis refers to the process of exact chromosomal duplication. The term *cytokinesis* is applied to the segmentation and division of the cytoplasm and to the end of cell division. Several stages of mitosis are distinguished conventionally: interphase, prophase, metaphase, anaphase and telophase.

In the *interphase* (Figure 2), the chromosomes are usually resolved into amorphous strands and masses of darkly staining chromatin in which it is hard to recognize individual chromosomes. Nevertheless, in some plant material it is possible to identify them individually, and in rare instances it is possible to detect them even as duplicated. The chromatin filaments are thought to represent the chromosomes in a state of swelling and uncoiling (*chromonemata*). In Figure 10C, the chromosome is shown to consist of two spiral coils that are thought to represent the chromonemata into which the chromosome dissolves during interphase.

The *prophase* is characterized by a condensation of the chromatin into recognizable thin, double, spiral filaments or *chromatids*. It may be preceded by a division of the centrosome, which is situated close to the nucleus, each of its halves containing a centriole. The centrioles move to opposite poles of the nucleus and initiate aster formation. As prophase progresses, the chromatids shorten and become thicker. Figure 10B shows the late prophase-early metaphase chromosome to consist of a pair of chromatids held together by a clear zone, the *centromere*. At this stage and during metaphase, the chromosomes may be classified according to the position of the centromere as *acrocentric*, *metacentric* and *submetacentric* (Figure 10A). Finally, during the late prophase, the nuclear membrane disintegrates, and the spindle appears almost complete.

During *metaphase*, the chromatid pairs line up equatorially across the spindle in a ring fashion. It can be seen that some of the fibers attach to the centromeres (so-called chromosomic fibers, as contrasted with the continuous fibers running from pole to pole without interruption).

Anaphase is preceded by a division of the centromeres, and is followed by a separation of the chromatids (now called daughter chromosomes) and their migration to the opposite poles of the spindle.

In *telophase* the chromosomes have reached their destination and are starting the process of prophase in reverse again, i.e., resolution into chromonemata and restitution of the nuclear membrane, followed by cytokinesis, with final disappearance of the spindle. Figure 11 reviews the chromosomal mechanisms during mitosis, in a diagrammatic fashion. The photos of the stages of mitosis of the whitefish blastula (Figures 2-9) are the most beautiful material that can be found to illustrate mitosis.

Recent refinements in tissue culture technics and cytologic methods have made it possible to study human chromosomes far better than ever before. There has been a consequent revolution in human cytogenetics, which has swept away, among other things, the old delusion that man had 48 chromosomes. It is now universally recognized that man has 46 chromosomes—22 pairs of autosomes and one pair of sex chromosomes, XX in the female

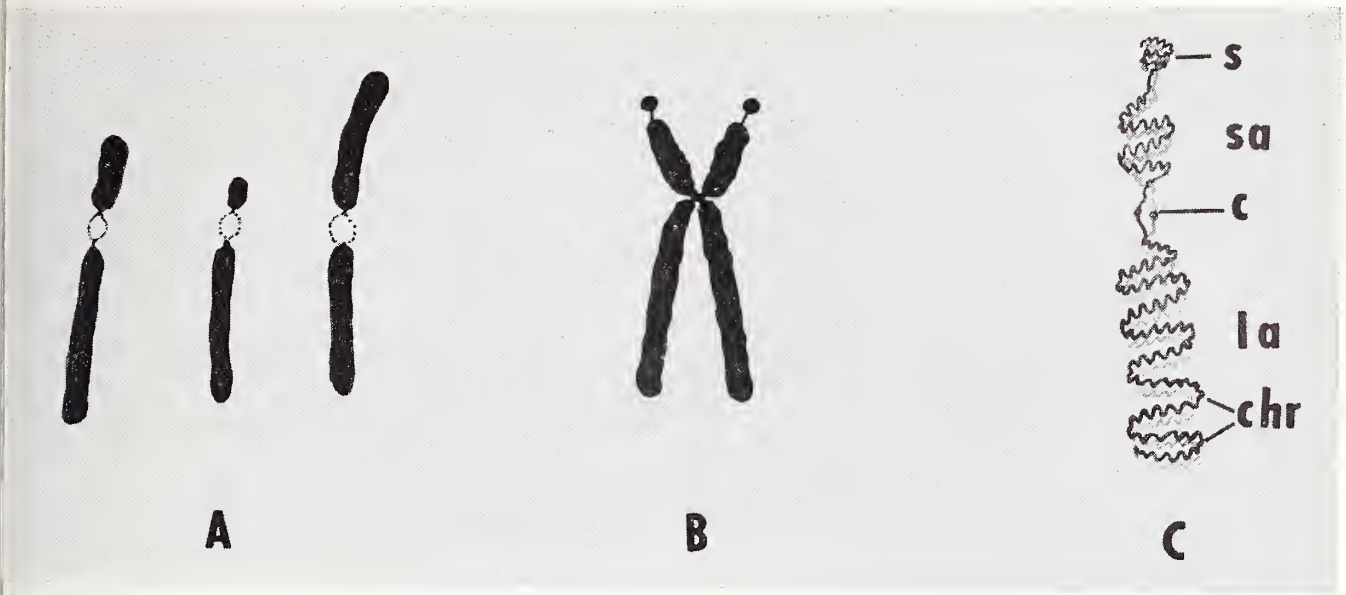


Figure 10. (A) Three types of chromosomes classified according to position of the centromere. Left to right: submetacentric, acrocentric and mediocentric. (B) Human metaphase chromosome. Two chromatids are held together by the centromere in a submetacentric position, resulting in the short and long arms. Satellites are also shown. (C) Internal structure of chromosome, showing two chromonemata fibers, as well as the major and minor coils. These can be visualized best after treatment with potassium cyanide. Symbols: s = satellite; sa = short arm; c = centromere; la = long arm; chr = chromonemata.

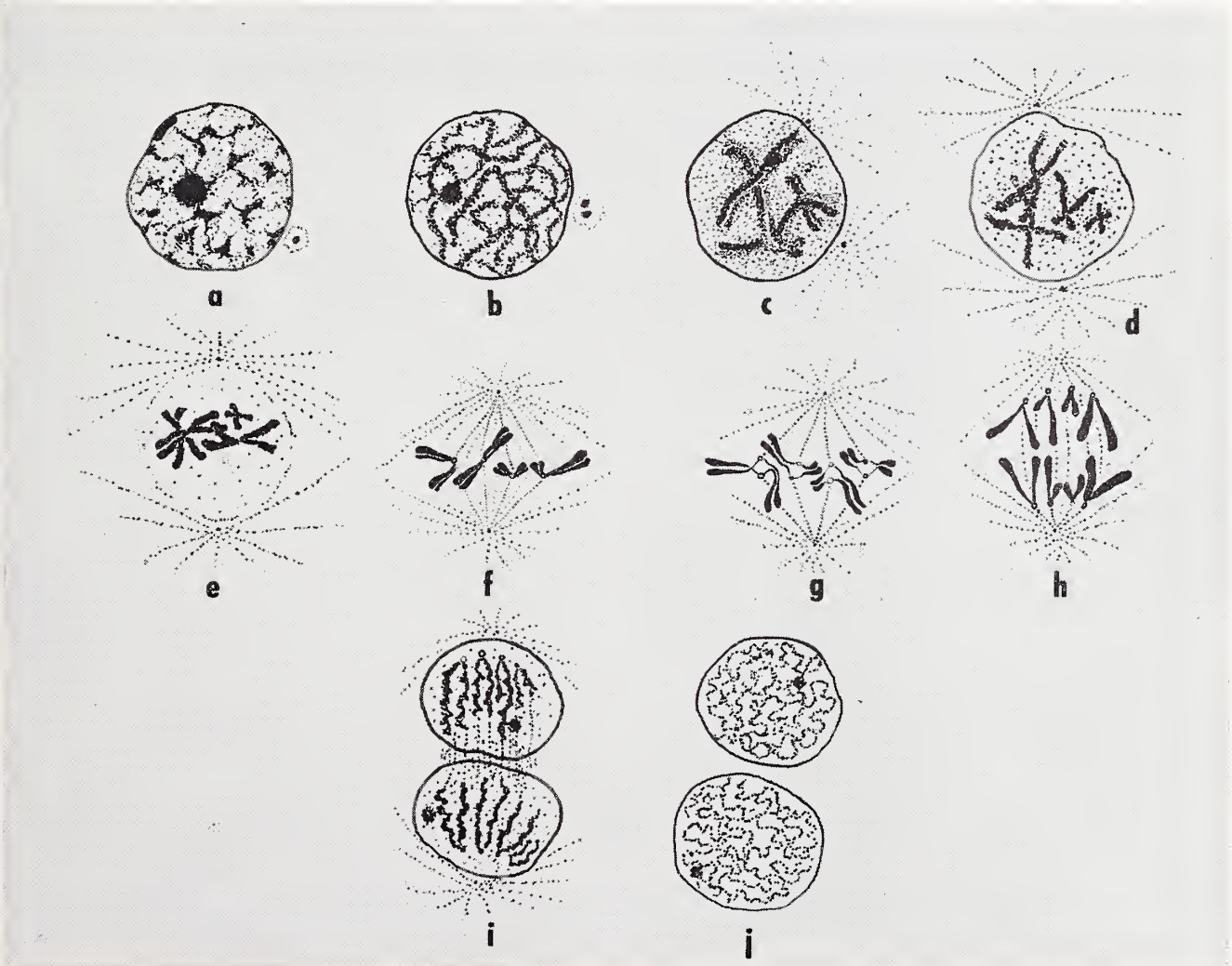


Figure 11. Diagrammatic résumé of mitosis. Cytoplasm has been left out. Symbols: a = interphase nucleus with nucleolus and sex-chromatin body; b and c = early prophase with condensing chromonema spirals; d = middle prophase with recognizable chromatids and asters; e = late prophase, disintegration of the nuclear membrane; f = metaphase, equatorial distribution of chromatid pairs; g = late metaphase-early anaphase division of centromeres; h = anaphase; i and j = telophase.

and XY in the male. By now, many chromosomal aberrations have been reported, the most important of them being the trisomy of one of the smallest autosomes (belonging to pair 21 or pair 22) characterizing mongolism, and the many sex-chromosomal anomalies described in the Turner and Klinefelter syndromes and their variants.

Figure 12A is the karyogram of a normal woman, and Figure 12B represents the idiogram of her husband. In the latter picture, the chromosomes have been cut out and arranged according to size and position of the centromere. Identification of the individual chromosomes is as yet hazardous (Patau).

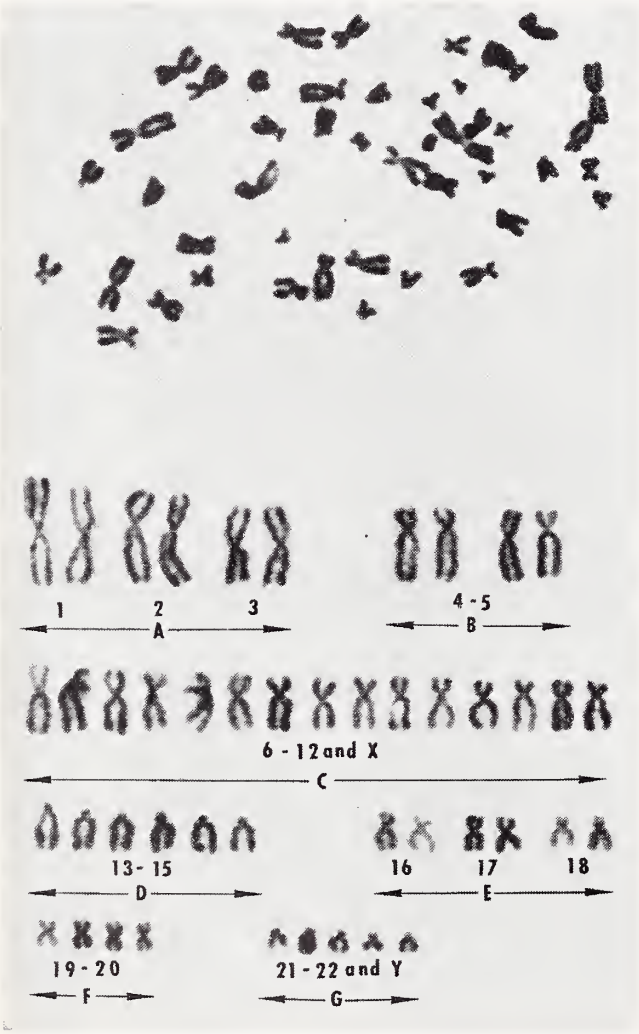


Figure 12. (A) Karyogram of a normal human female. Metaphase chromatid pairs number 23 (2n = 46). (B) Idiogram of her normal husband. Cells are arranged according to the method of Patau. Pairs that can be identified with certainty are numbered, and the rest are arranged in the usual scheme, in order of decreasing size, in seven groups. Ferguson-Smith is able to identify satellites on at least five pairs: 13, 14, 15, 21 and 22. The two X chromosomes cannot be identified with certainty, but the Denver convention places the X between pairs 6 and 7. Thus, in the male, there are 15 chromosomes (14A + X) in group C, and five chromosomes (4A + Y) in group G. In the female, there would be 16 chromosomes in group C (14A + XX), and only four in group G.

MEIOSIS

Meiosis is a special form of mitosis that occurs during the formation of germ cells. Three important events take place during meiosis:

1. Reduction of the chromosome complement to the haploid state
2. Free segregation of the chromosomes into the gametes
3. Crossing-over.

Reduction of the chromosome number is a complex process, but very necessary in order to avoid continual doubling of chromosomes with every generation. Figures 13-19 are photomicrographs of the stages of meiosis in the male from squash preparations of the testis of the locust. Figures 20-22 illustrate the process of polar-body formation during ovogenesis of the round worm.

Roughly, there are five stages in the prophase of the first meiotic division:

Leptonema. During this stage, the chromosomes condense and form loops equal in number to the diploid number of the species ($2n$). They frequently arrange themselves in a bouquet-like fashion.

Zygonema. This is the stage of pairing of homologous chromosomes, which approach and approximate by pairing point by point, producing n numbers of chromosome loops (Figure 13).

Pachynema. Following the pairing, the chromosomes contract and divide longitudinally so that there are a haploid number of tetrads composed of four chromatids (Figure 14).

Diplonema. Now, the homologous portions start to separate, retaining attachments by their points of crossing-over, the *chiasmata* (Figure 15A).

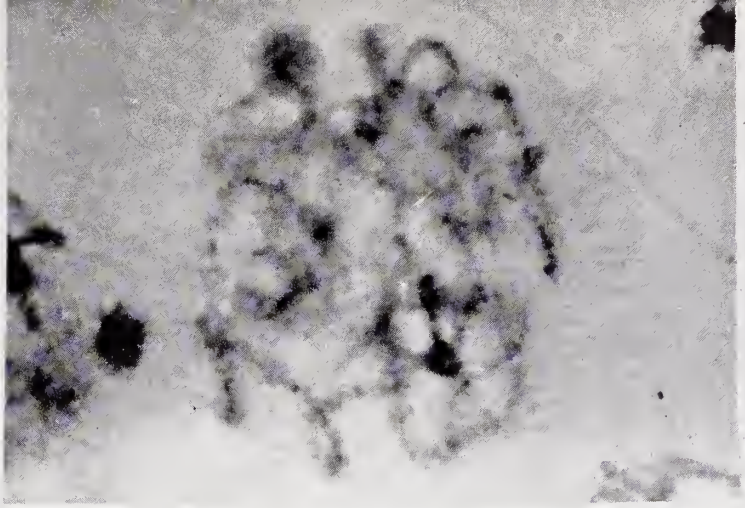
Diakinesis. This is characterized by further contraction of the chromosomes, with a disappearance of the splitting of the diplotene stage (Figures 15B and 16).

Thus we notice a fundamental difference between mitosis and meiosis. In the former, chromosomes do not conjugate, and crossing-over occurs only during meiosis. The prophase stages are identical in spermatogenesis and ovogenesis.

Next follows metaphase I, during which the tetrads separate equally, forming two cells each containing $2n$ pairs of chromatids which will split equally during the second meiotic division (Figure 17), resulting in four cells with a haploid chromosome complement, each cell completely different from the others. In Figure 23 the process is illustrated diagrammatically. Please observe the possible alternate arrangement of the pairs of

→

Figures 13-19. Meiosis and spermatogenesis in the grasshopper testis. Figure 13. Zygotene. Figure 14. Pachytene; X = X chromosome. Figure 15. A = diplotene; B = early diakinesis. Figure 16. Diakinesis with 12 tetrads. Figure 17. Polar view of metaphase II; 12 chromosomes. Figure 18. Anaphase II, single chromosomes. Symbol: s = spermatozoa. Figure 19. Group of eight spermatids.



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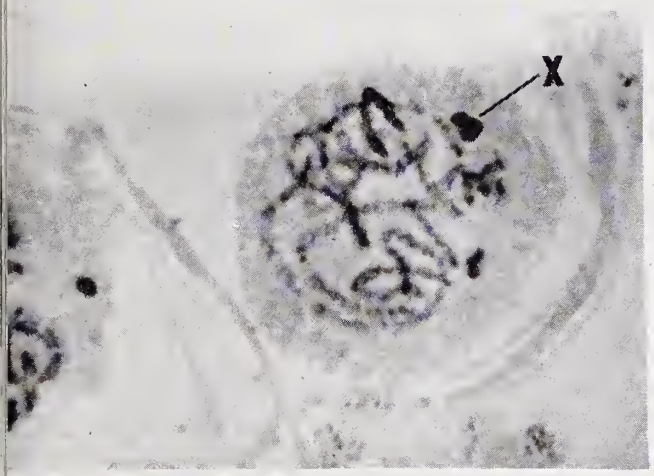

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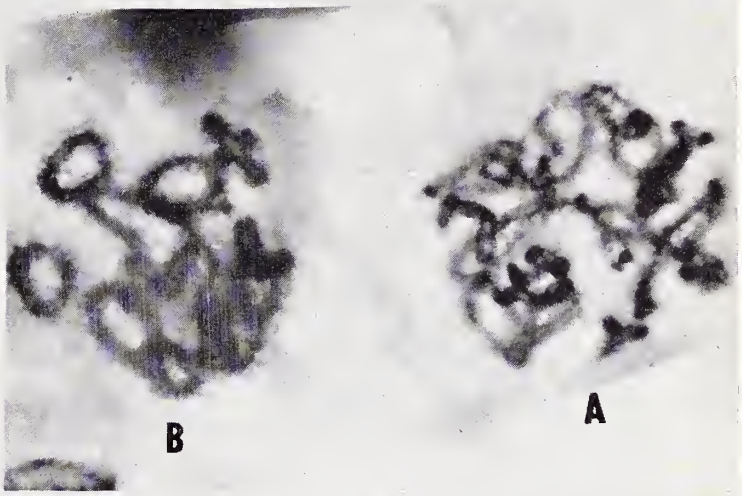

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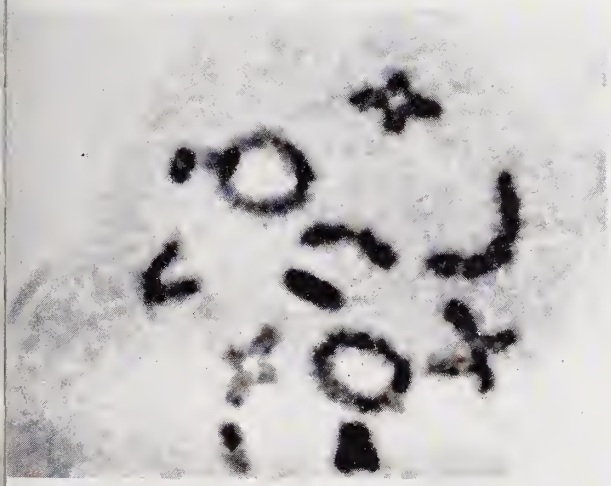

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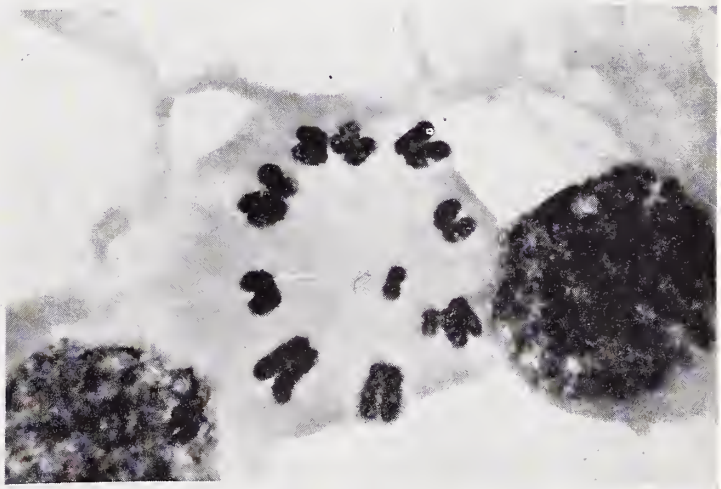

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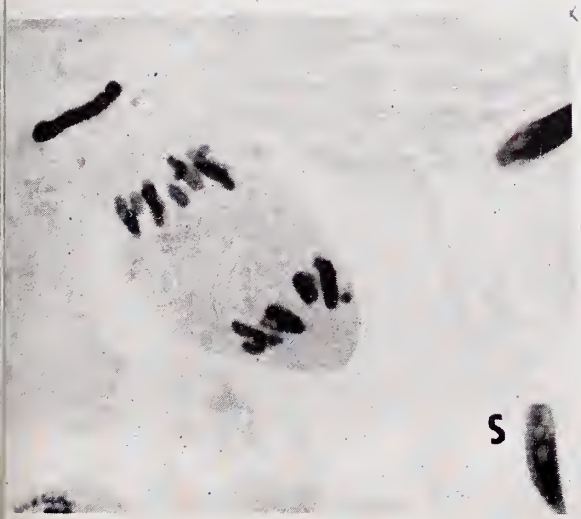

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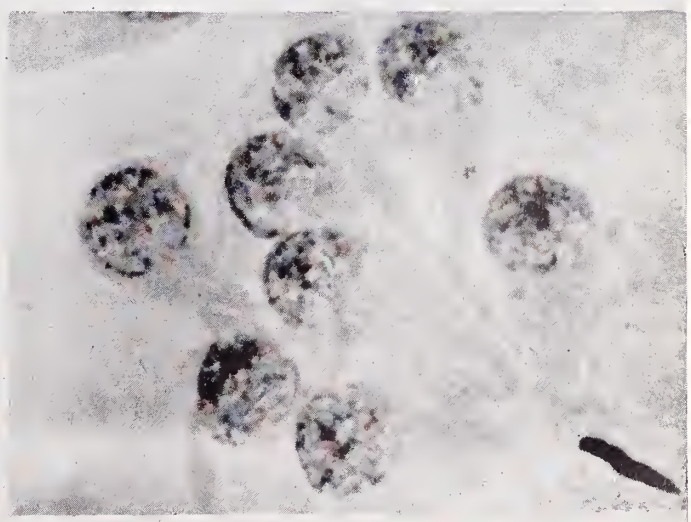


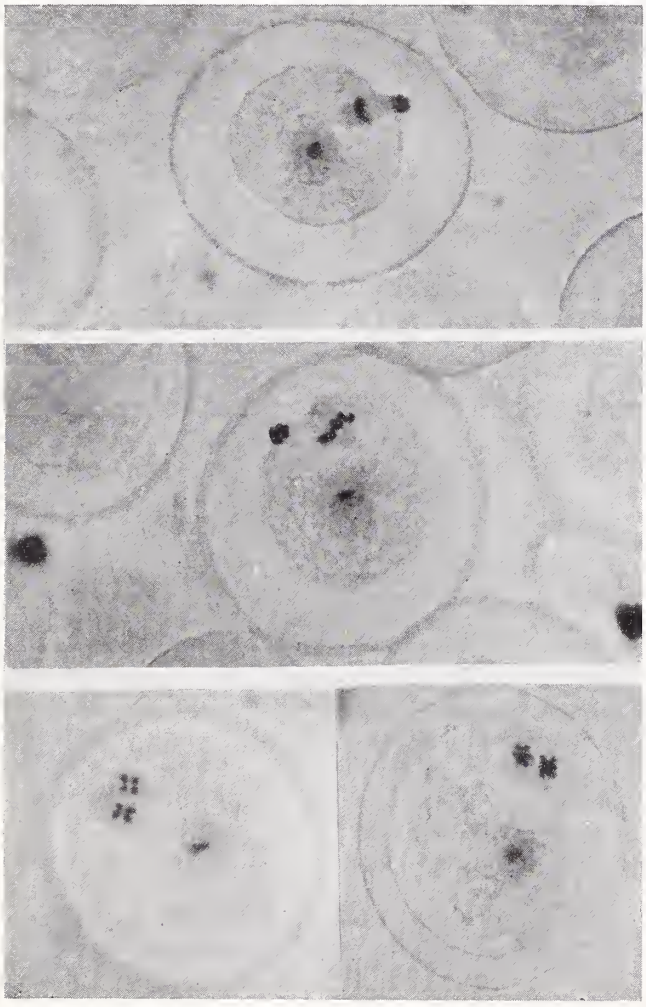
19

homologues on the spindle. Thus, four different arrangements are possible in this example (n = the number of pairs of chromosomes, and the number of kinds of gametes = 2^n). In man, with 23 pairs, the number of possibles is 2^{23}, or 8,388,608 (Stern). In this way it is possible to achieve enough diversity and genetic heterogeneity to assure each offspring's having a totally individual character.

CROSSING-OVER

In Figure 23, it is seen that the two tetrads each have one chiasma—one place of crossing-over—resulting in a reciprocal exchange of portions of homologous chromosomes and a rematching of groups of genes with different alleles. Rearrangement of the chromosomes is another means of achieving genetic diversity. In Figure 24, crossing-over is illustrated diagrammatically for one pair of homologous chromosomes with only one point of crossing-over. However, crossing-over may occur in two, three or even more places along the chromosomes. The difference is seen in the types of gametes produced if crossing-over does not occur.

In 1919, T. H. Morgan encountered the following situation. Breeding homozygous dominant fruit flies with gray bodies and long wings (*GLGL*) and the doubly recessive type with black bodies and vestigial wings (*glgl*), he obtained only gray, long-winged flies (*GLgl*). However, from the test cross of the F-1 flies with the recessive male parent (*GLgl* x *glgl*), he obtained not only 83 per cent gray, long-winged (*GLgl*) and black flies with vestigial wings (*glgl*) in equal numbers (showing linkage between the two pairs of genes *GL* and *gl*), but also an unexpected 13 per cent of gray flies with vestigial wings (*Glgl*) and of black, long-winged flies (*glgl*). This discovery, along with others that were made in quick succession, demonstrated that linkages are not always constant, but are broken with a certain frequency characteristic for each pair of genes under study. (Please refer again to Figure 24.) By observing accurately the linkage and cross-over values of many genes, Morgan was able to establish a linear



Figures 20-22. Polar-body formation in ovogenesis of Ascaris. Figure 20 (top) shows formation of the first polar body. Figure 21 (center) shows formation of the second polar body. Figure 22 (bottom) consists of two views of the ovum with the two polar bodies. The first polar body did not divide again.

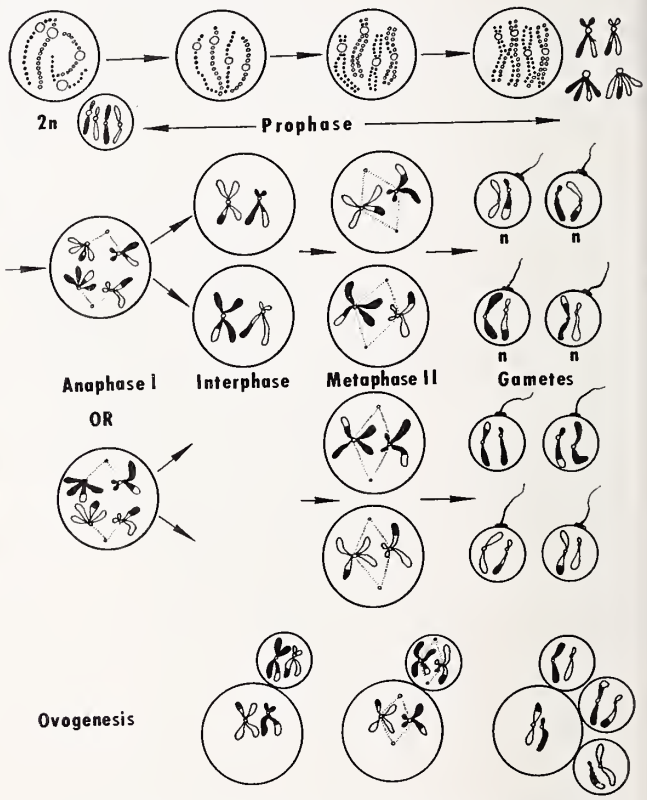


Figure 23. Diagrammatic representation of meiosis. Two pairs of chromosomes (2n = 4), one mediocentric and one sub-mediocentric. One member of each pair is white, and the other is black. There is one point of crossing-over for each pair. The two possibilities of reduction of the tetrads are shown. The process is illustrative of spermatogenesis yielding four equal cells. Ovogenesis is shown only after formation of the first polar body. Here again, two choices of reduction are possible but are not shown.

order of genes on the chromosomes, and he received a Nobel prize for his work.

This process is illustrated by the following example of "linkage group number two" (i.e., chromosome II) in Drosophila melanogaster:

1. White eyes and silver body have a cross-over value of 1.5 per cent.

2. Silver body and ruby eyes have a cross-over value of 7.5 per cent.

3. Ruby eyes and white eyes have a cross-over value of 6.0 per cent. From these findings it is evident that the order of genes is silver-white-ruby. Thus, a low cross-over value such as 1 or 2 per cent signifies close proximity of the two genes. A cross-over value of 50 per cent means complete randomness of crossing-over, or very weak linkage—i.e., residence at opposite ends of the chromosome.

In this manner, Morgan and his coworkers established maps for all the chromosomes of the fruit fly D. melanogaster. Similar chromosome maps have been constructed for other insect species and for some plants. In mammals, the work has progressed very slowly. The only autosomal linkages known in man to date are the Lutheran: Lewis red cell phenotypes; elliptocytosis: rhesus loci; and the nail-patella syndrome: ABO alleles. There are some less well substantiated linkages and many sex-linked conditions in man. As for the latter, the genes are located on the heterologous portion of the X chromosome. Holandric inheritance, in which the genes are located on the heterologous segment of the Y chromosome, has been described repeatedly, but Stern has directed criticism at the more classic examples. Morton, in a review of partial sex linkage, found little evidence for any locus studied in man, with the possible exception of the syndrome of hereditary nephritis and deafness. Even though Ford reaffirms the possibility of crossing-over in the homologous segments of human sex chromosomes, there are others who doubt its occurrence in man (Matthey). For these reasons, we can offer only a partial table of sex-linked conditions, instead of the classic picture of the sex chromosomes in man that was constructed by Haldane (Table 1).

TABLE 1

PARTIAL LIST OF COMPLETELY X-LINKED CONDITIONS

Recessive:
- Hemophilia (AHG deficiency)
- Christmas disease (PTC deficiency)
- Partial color-blindness
  - a. Protanomaly dominant to protanopia
  - b. Deuteranomaly dominant to deuteranopia
- Aldrich's disease
- Progressive muscular dystrophy (Duchenne)
- Myopic night-blindness
- Sex linked optic atrophy

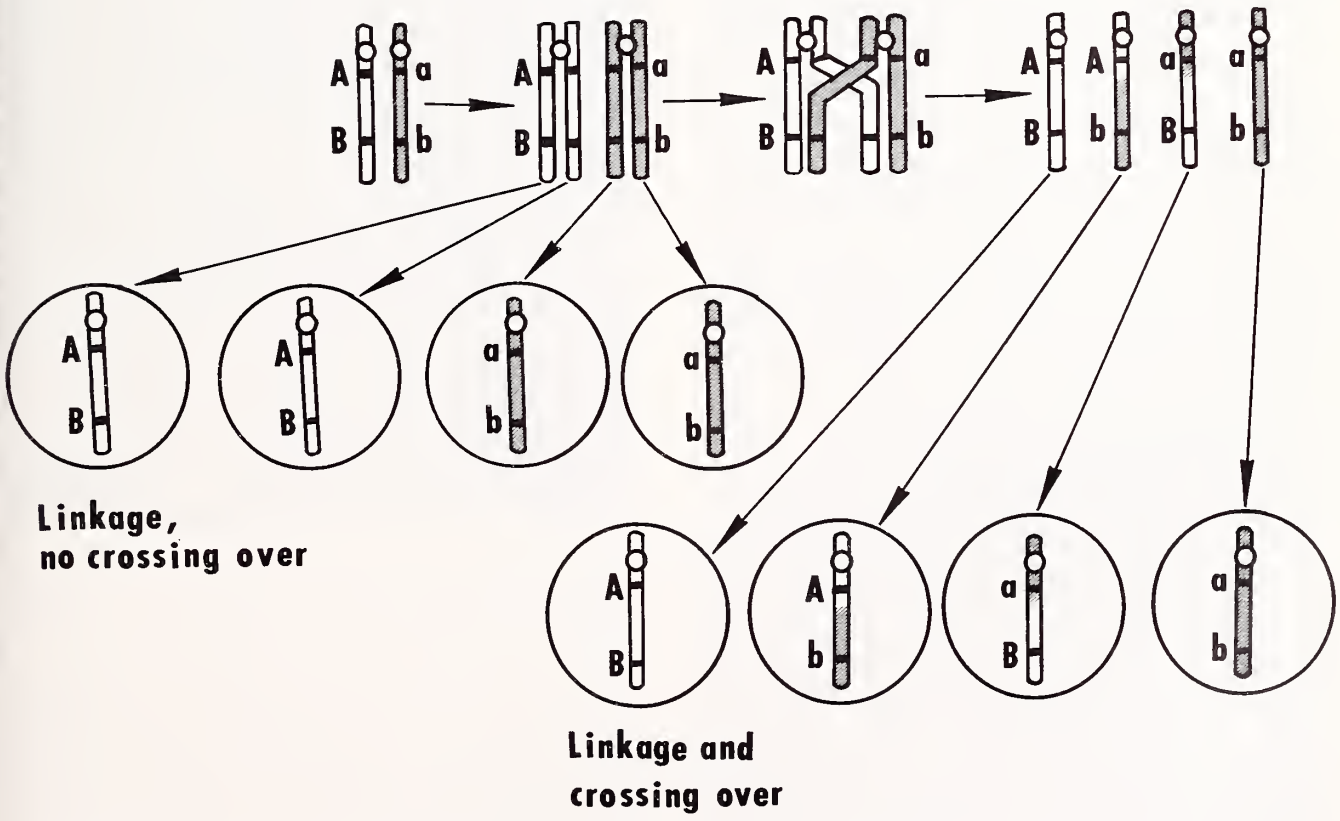


Figure 24. Diagram of linkage, crossing-over, and the possibilities of segregation with and without crossing-over.

Sex linked Leber's disease
Sex linked ectodermal dysplasia
Sex linked ichthyosis
Agammaglobulinemia
Sex linked diabetes insipidus (renal)
Sex linked ADH-deficient diabetes insipidus
Ocular albinism
Inability to smell HCN
Total color-blindness (Pedigree 9 in Part I of this review)
Sex linked Hurler's disease
Pseudoglioma with mental retardation
Hypomaturation of enamel
Angiokeratoma diffusum corporis universale (Fabry)
Keratosis follicularis spinulosa cum ophiasi
Hypochromic, microcytic anemia
Retinal detachment
Microphthalmia
External ophthalmoplegia and myopia
Nystagmus
Sex linked type of Charcot-Marie-Tooth peroneal muscular atrophy
Sex linked spinal ataxia
Sex linked spastic paraplegia
Sex linked cerebellar ataxia
Progressive deafness
Oculo-cerebro-renal syndrome of Lowe
Megalocornea
Hypoparathyroidism
Hydrocephalus dueto, congenital stenosis of aqueduct of Sylvius

Intermediate:
Choroideremia
Tendency to favism (glucose-6-phosphate dehydrogenase deficiency)

Dominant:
Enamel hypoplasia
Vitamin D resistant rickets (hypophosphatemia)

THE CHROMOSOME AND DNA

In the interphase nucleus (Figures 25 and 26), there is little structural evidence of the complex and dynamic aspect of genetic coding and transmission of information. In an effort to get at the basic hereditary molecules of the nucleus, two main approaches have been used—the biochemical and the cytological, using cytochemical and photometric absorption methods. From various fractionating experiments, two important nucleoproteins have been isolated: desoxyribonucleic acid (DNA), and ribonucleic acid (RNA). In the whole chromosome, DNA constitutes 90-95 per cent of the nucleic acid. Ordinarily, DNA and RNA may be differentiated by the fact that DNA reacts positively with diphenylamine and the Feulgen stain, and RNA with orcinol and basophilic dyes after ribonuclease treatment.

It is now generally accepted that DNA (Figure 29) is the main carrier of our genetic material, and that RNA is intimately concerned with the synthesis of proteins, acting in a transmitting fashion

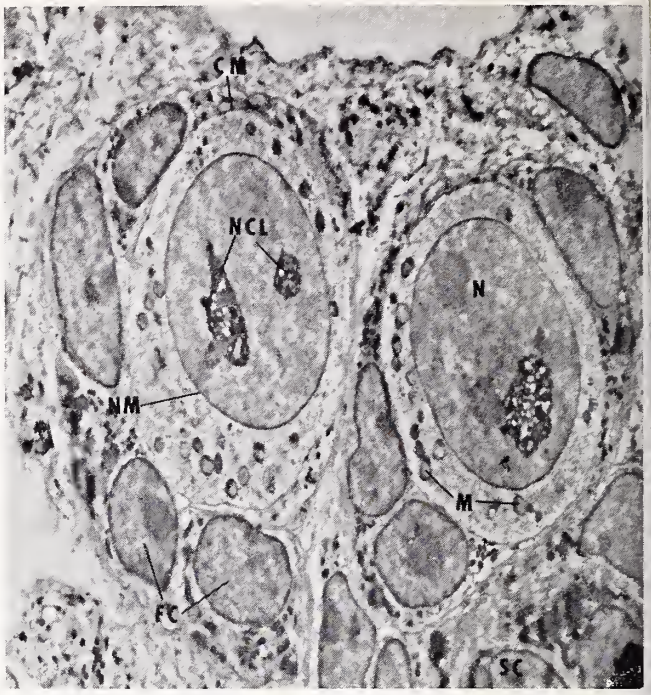


Figure 25. Electron-photomicrograph of two guinea-pig primary oogonial follicles (x7000). CM = cell membrane; N = nucleus; NCL = nucleolus; M = mitochondria; FC = follicle cells; SC = stromal cells; NM = nuclear membrane.

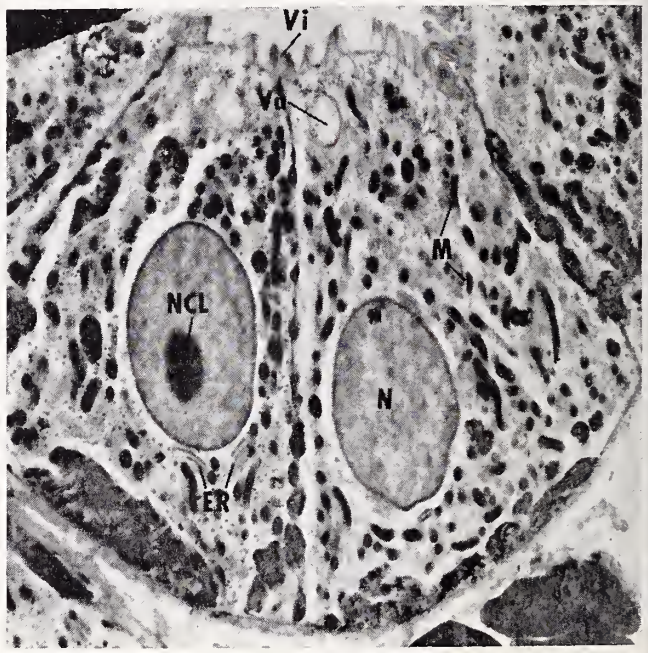


Figure 26. Electron-photomicrograph of two cells of the proximal tubule of the kidney of the horned toad (Phrynosoma cornutum) (x3000). Symbols as in Figure 25, plus ER = endoplasmic reticulum; Va = vacuole; and Vi = microvilli.

by carrying the coded information from the DNA of the nucleus to the ribosomes of the cytoplasm, where protein synthesis is thought to occur, using another form of RNA as a template. Investigations concerning the structure of DNA have culminated in the model constructed by J. D. Watson

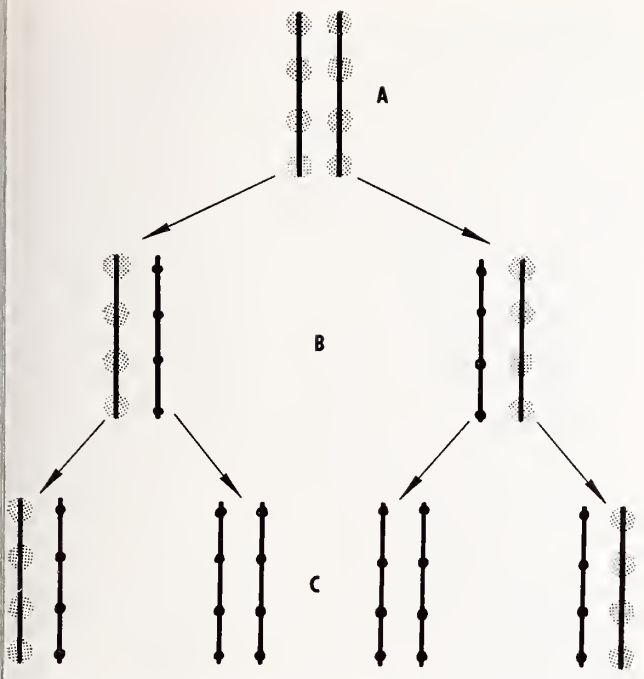


Figure 27. Diagrammatic illustration of experiments of Taylor on the incorporation and subsequent fate of H_3-T into DNA (see text). A = parent DNA, both chromatids labelled; B = first generation chromatids, only one pair labelled; C = second generation DNA, out of four chromatids only one labelled.

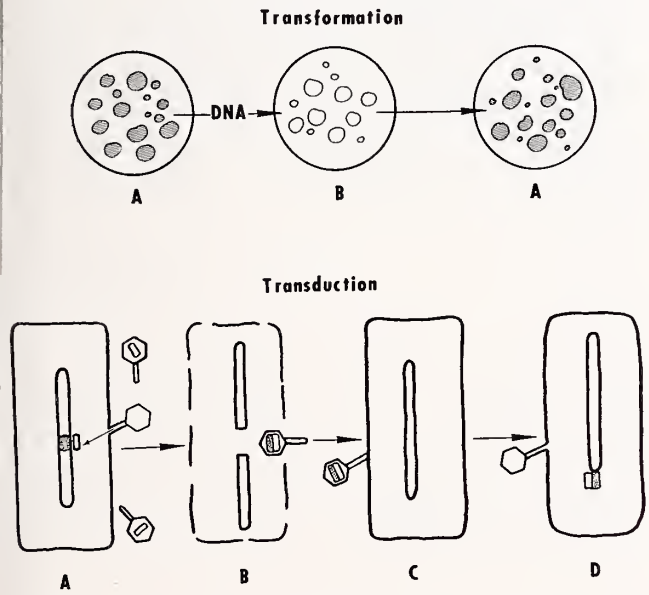


Figure 28. The top drawings show the transformation of pneumococcal strains by DNA. A = penicillin-resistant strain; B = penicillin-susceptible type. The bottom drawings sketch the transduction experiment of Zinder and Lederberg. A. Virus has adsorbed on cell surface and injects its DNA into the bacterium, where it associates with the bacterial "chromosome." B. Bacterium is lysed. A virus containing a particle of host-cell genetic material is taken to a second strain. C. Second strain not possessing the trait of the first bacterial strain in A. D. The particle attaches to the cell's genetic material and endows it with the transmitted trait.

and F. C. P. Crick, as yet controversial in many aspects but to date the best available model to fit the theoretical requirements and to explain most of the phenomena that have been observed. Evidence that DNA is indeed associated with the chromosomes and performs genetic functions accumulated slowly, but presently there exists absolutely no doubt about the association.

In the first place, the presence of DNA in the chromosomes was demonstrated by the above-mentioned staining methods, as well as by the photometric absorption technics. These have conclusively established the ubiquity of DNA and the association of DNA and chromosomes. Secondly, it has been shown that the nucleus contains DNA in amounts proportional to the number of chromosomes, i.e., the haploid nucleus (n) has one-half the amount of DNA possessed by the diploid ($2n$), which in turn has one-fourth as much as an octoploid ($8n$) nucleus. These studies also correlate with the nuclear size, e.g., the greater its DNA content, the larger is the nucleus. Coincidentally, it was also discovered that DNA does not duplicate during the prophase, but halfway through interphase. Thirdly, according to the Watson-Crick model, DNA consists of two helical spirals. The unwinding of the two parent polynucleotides occurs at the same time that duplication takes place. The experiments by Taylor in plants confirmed this theory. He incubated plant cells in tissue culture media with Tritium-labelled thymidine (H3-T), chosen because the base thymidine is present only in DNA. After the incorporation of H3-T into the DNA, cells are then transferred to normal, non-radioactive medium, and mitosis is interrupted with colchicine, which however does not prevent chromosome duplication, thus allowing the retention of the new first- and second-generation chromosomes in the parent cells. It is then found that radioactivity is equally distributed between chromatids of each first-generation chromosome. However, in the second chromosome generation, synthesized in normal medium, only one of the chromatids is found to be labelled (Figure 27).

EVIDENCE FROM BACTERIA AND VIRUSES

Direct and indirect evidence concerning the nature and function of DNA has accumulated from bacterial and viral studies over the last few years. This includes the classic demonstrations, of transformation, transduction and conjugation in bacteria, culminating in the Nobel Prize winning work of Lederberg.

In 1944, Avery, McLeod and McCarty identified DNA as the substance responsible for *transformation,* a phenomenon in which certain characteristics of one strain of pneumococci, such as streptomycin or penicillin resistance or mannitol utilization, may be transferred to another strain of pneumococci without these traits, by inoculating the latter with (DNA containing) alcohol extracts of

the former. Just how the DNA of the donor organism becomes incorporated into the genetic make-up of the recipient is unknown (Figure 28A).

Phages are viruses that infect bacteria. Virulent strains bring about a cessation of protein production in the infected bacterium and cause it to replicate the DNA which the virus had injected. Then new capsules are made for the viral DNA, and the host cell is destroyed, liberating a large number of new phages. In temperate strains, the injected DNA becomes associated with the genetic structure of the bacterium in some way, allowing for its duplication synchronously with the replication of the bacterial chromosome. This is the state of *lysogeny,* and the phage in this stage is called a *prophage.* The lysogenic state is reversible; the prophage may be activated, leading to lysis of the cell. Studying temperate phage lysis in Salmonella, Zinder and Lederberg discovered, in 1952, that the phage carries away a particle of the original bacterial chromosome to a new host, leading to the permanent acquisition of the characteristics of the original bacterium (Figure 28B).

Lack of space forbids me to discuss the complex process of conjugation in bacteria, but I should like to mention a recent brilliant experiment by Novelli demonstrating gene action outside of the living cell. He used several strains of E. coli, specifically the types which produce the enzyme beta-galactosidase, a mutant lacking this trait, and a strain with a suppressor gene that induces the formation of the enzyme only when pretreated with a suitable inducer.

He prepared a cell- and DNA-free E. coli soup, taking care to preserve the ribosomes that synthesize the proteins. To this mixture, he added, among other compounds, amino acid building blocks for the enzyme and nucleotides of messenger RNA. This mixture produces beta-galactosidase, following the addition of either DNA from the strain which regularly synthesizes the enzyme, or from an inducible strain that had been pretreated before its DNA was extracted. The enzyme was also produced by the addition of DNA fragments of as yet undetermined size. Adding DNA from strains that do not produce the enzyme failed to induce formation of beta-galactosidase in the mixture, and the addition of DNA from suppressor strains which had not been pre-treated inhibited further enzyme synthesis immediately.

THE GENE

Demerec recently reviewed the physical aspects of the gene, collating the work of others and his own experience with Salmonella typhimurum mutants. He stated the length of the gene at 0.68 microns—a surprisingly large size. Thus, if the correct number of genes for Drosophila melanogaster is 2,000, Bridges was probably right in assuming that a gene represents several bands of

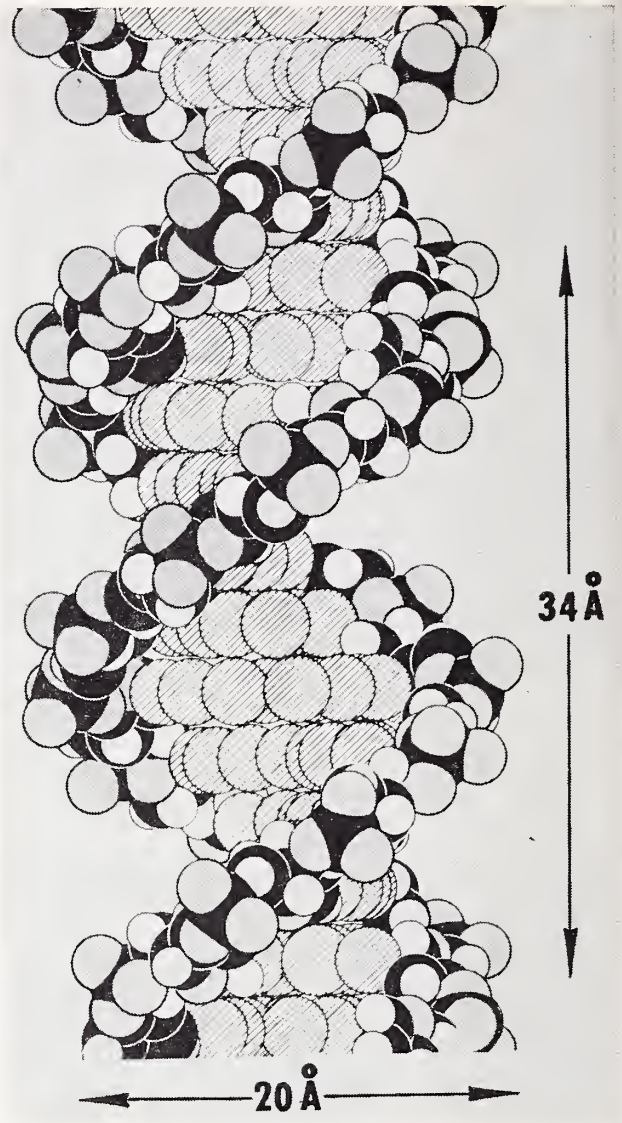


Figure 29. Courtesy: L. D. Hamilton, after Feughelman et al.[29] A schematic representation of a portion of the DNA molecule in the B, or paracrystalline, form. This is based on the model devised by J. D. Watson and F. H. C. Crick, of Cambridge, England, reflecting biochemical and diffraction data as well as some logical assumptions. This illustration shows clearly the ropelike nature of the molecule, consisting of two desoxyribosephosphate chains wound about each other in a double helix, and united by closely fitting pairs of nitrogenous bases forming shelf-like layers of molecules represented by the larger, shaded circles.

The chains consist of phosphates linking together pentose sugars to which are attached in a repetitious sequence the bases adenine, cytosine, guanine and thymine. The two chains are loosely united by hydrogen bonds between the bases adenine:thymine and cytosine:guanine in an alternating fashion.

Species differences affect only the type of purine or pyrimidine base. The basic molecular arrangement is identical for all species examined so far.

The molecule is 20 Å thick, but at least 1,000 times as long.

the polytenic salivary gland chromosomes. Nevertheless, the diameter of the two strands of DNA is only 20 Ångstroms—far too low to be resolved by the light microscope. This length of the gene would be equivalent to 2,000 nucleotide pairs.

It has been demonstrated in microorganisms that many mutations may occur per gene. Demerec has 4,000 S. typhimurum mutants in his laboratory, representing only 60 gene loci at most. Therefore the gene loci are complex in structure and contain many "*sites.*" A maximum of 2,000 sites have been calculated per locus, and thus a site probably represents a single nucleotide pair.

The finest demonstration of the specificity of effect of nucleotide alteration, as well as of the 1:1 relationship between gene and protein structure, was discovered by Ingram (1957) in sickle cell anemia, a recessively inherited hemolytic anemia occurring predominantly in Negroes. It was found that hemoglobin S ("sickle") varied from normal adult (Hgb *A*) hemoglobin by a single amino acid. Valine had been substituted for glutamic acid. Similarly, hemoglobin *C* differs from hemoglobin *A* by the raplacement of lysine for glutamic acid in exactly the same position.

SUMMARY

In this short survey of basic genetics, I have reviewed the brief (100 yr.) but extraordinarily momentous development of the science, from the basic discoveries of Mendel to the complexities of genetic cytochemistry today. I have attempted to bridge the gap between the clinical picture and the gene by presenting a selection of the most important research concerning the basic aspects of human heredity, cytogenetics, and bacterial and viral genetics. There is no doubt that DNA represents the carrier of genetic information. Details of its structure and function, however, will require further elucidation.

ACKNOWLEDGMENTS

I wish to thank Mr. T. Rey, of the Upjohn Company, for permission to reproduce the magnificent series of photomicrographs of mitosis, and also Figure 29, a diagram of the DNA molecule. For the latter illustration, I owe additional thanks to Dr. Leonard Hamilton, of the Sloan-Kettering Institute, who originally devised this method of reproducing DNA.

I am exceedingly grateful to Dr. G. Brosseau, of the S.U.I. Zoology Department, for permission to use his photos of oogenesis of ascaris and to photograph his squash preparation of grasshopper testes, as well as for his invaluable help in identifying the stages of meiosis.

I also appreciate the kindness of Dr. R. G. Bunge, of the Department of Urology at S.U.I., in helping me to take photomicrographs of grasshopper spermatogenesis with his micro-photo apparatus.

My thanks also go to Professor Hans Zellweger and to Mr. K. Mikamo, of the Chromosome Laboratory in the Department of Pediatrics at S.U.I., for permission to use the two photomicrographs of normal male and female human chromosomes.

Finally, I wish to thank Dr. E. Anderson, of the S.U.I. Department of Zoology, for supplying me the two magnificent electron-photomicrographs that have been reproduced here as Figures 25 and 26.

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Clinicopathologic Conference

Mercy Hospital, Des Moines

INFANT V, A MALE, was born at Mercy Hospital, Des Moines, on April 13, 1961, at 4:33 p.m. He expired on April 14, 1961, at 4:28 a.m.

Maternal History: Mrs. K. A. V., a 33-year-old white female, had been admitted to Mercy Hospital, Des Moines, at 8:50 a.m. on April 13, 1961. She was gravida v, para ii. She gave a history of one pregnancy lasting approximately seven months. The death of a premature baby occurred 48 hours after birth. The cause of death was not stated. There had also been one miscarriage during the third month of pregnancy. There were two healthy living children.

Family History: The family medical history and maternal medical history were noncontributory.

Natal History: The present pregnancy had apparently been uncomplicated until Mrs. V. went into labor in the thirty-fourth week of pregnancy and was admitted to the hospital at 8:50 a.m. on April 13. The membranes were ruptured artificially at 9:47 a.m. on that day. Analgesia during the first stage consisted of 100 mg. of Demerol. At 2:30 p.m., during the first stage of labor, the patient developed a sudden lower-abdominal pain, followed by the passage of dark blood from the vagina. The fetal heart sounds at that time were not audible. The bleeding continued, although at a diminished rate. At 4:00 p.m. the heart tones were heard at a rate of approximately 100 per minute. Bloody fluid was still exuding from the vagina. At 4:15 p.m. the fetal heart rate was 120 per minute. The cervix was fully dilated. Station was plus 3 in the right occiput anterior position. Dilatation was complete at 4:20 p.m. The second stage of labor occurred at 4:30 p.m., April 13. Spontaneous delivery of a viable male infant was accomplished with a midline episiotomy. The baby was flaccid and pale, and his respirations were poor. The heart rate was 60 to 70 per minute. The baby did not cry immediately following birth.

The placenta and membranes were delivered at 4:55 p.m., April 13. Examination of the placenta revealed a velamentous insertion of the umbilical cord, with rupture of a vessel. The estimated blood loss during labor was 250 cc.

The total duration of labor was 8 hrs. and 35 min. Resuscitative measures were carried out, and approximately 10 cc. of secretions were obtained by tracheal suction. The baby was transferred to the nursery, breathing slowly. The lungs were adjudged incompletely expanded. The heart rate was 96 per minute.

Clinical Course: The infant's weight was 6 lb. 4 oz. The clinical impression was that of exsanguination from a ruptured umbilical vessel. At 5:20 p.m. the respirations were still of poor quality. The pulse rate was approximately 60 per minute. A transfusion of 150 cc. of type O, Rh negative blood was administered. When 120 cc. had been transfused, the infant's color became pink. The cardiac rate increased to 80 per minute. The respirations were still embarrassed, but the infant seemed less pronouncedly in shock. The liver was not palpably enlarged. The lungs were clear.

The heart rate was 98 per minute at 7:30 p.m. At 8:00 p.m., an additional 20 cc. of whole blood was administered. The systolic blood pressure was stated to be 110 mm. Hg, and the heart rate was 126 per minute. The color was good. Two cubic centimeters of calcium gluconate was administered. At 8:30 p.m. another 25 cc. of whole blood was administered. The blood pressure at that time was 80 mm. Hg, and the heart rate was 100 per minute. At 11:30 p.m. the infant was described as somewhat pale, but more active. The respirations were 30 per minute, and the pulse rate was 130. A hemoglobin at that time was 14.4 Gm.

At 2:30 a.m. on April 14, the infant started bleeding from the nose, rectum and penis. The hemoglobin fell to 11 Gm. per cent. A transfusion of fresh whole blood was to be given, but the infant expired at 4:28 a.m., April 14, at the age of 12 hours.

CLINICAL DISCUSSION

Dr. Jack Spevak, pediatrician: If I were to name the clinicopathologic conference this afternoon, its title would be "Posthemorrhagic Shock of the Premature Infant." This is something that doesn't happen very frequently, but occurs often enough so that it becomes a familiar syndrome to both the obstetrician and the pediatrician.

In my discussion of this case, I should like to deal with the problem of posthemorrhagic shock—its clinical picture and method of treatment—and also try to explain the cause for the generalized bleeding that this baby developed terminally.

The mother was a 33-year-old white woman, gravida v, para ii, who had had a previous mature baby that had died 48 hrs. after delivery. That baby's cause of death was not stated. She also had had a miscarriage. At the time of this pregnancy, she had two living, healthy children. Signs of difficulty appeared at 2:30 in the afternoon, when she was in the first stage of labor. She developed abdominal pain and began to have heavy bleeding from the vagina. At that time it was stated that "fetal heart tones were inaudible." During the next hour and a half, the bleeding continued, but to a lesser extent.

Here is a mother about to deliver a premature infant, and troubles arise. Then is the time for

the obstetrician to get busy and prepare for the complications that may take place in the baby. Exactly what they may be, he has no way of knowing. It may be a placenta previa or a placenta abruptio, and he may deliver an asphyxiated infant who has difficulty with its respiration as part of its prematurity. Or he may deliver a baby who bleeds at the time it is being born. The latter event took place in this particular case. Then is the time to alert the anesthetist, the pediatrician, the laboratory and the blood bank, in order that the infant may have the best possible chance for survival. This point must be emphasized, for it is important.

At 4:00 p.m., the heart tones were 100 per minute, and the mother still showed bloody flow. At 4:15 p.m., the infant's heart rate was 120 per minute, and the cervix was dilated. Delivery followed at 4:30 in the afternoon—two hours after the first indication of maternal difficulty.

The baby was flaccid and pale, and his respirations were embarrassed. The baby was in trouble. What was the trouble? It could have been asphyxia. It could have been a pulmonary problem, a distress syndrome such as atalectasis, or possibly it could have been due to hemorrhage. It seems that when a baby is born after its mother has run into difficulty of this type, all of us think too often about asphyxia and prematurity, and tend to forget that a premature baby is capable of bleeding. Sometimes a great deal of time is wasted in looking for causes other than hemorrhage. In this case, the placenta and membrane were delivered 25 minutes later, and an examination of the placenta revealed an abnormal insertion of the umbilical cord—namely, a velamentous insertion, with rupture of a blood vessel. So the diagnosis was established 25 minutes after the baby was born. It may not be so easy to establish a diagnosis when the bleeding has been occult, as is the case when the baby has bled into the mother.

The infant was examined, tracheal suction was carried out, and the infant was transferred to the nursery. It is stated that the lungs were not completely expanded, and the child had a heart rate of 96 per minute. The infant was said to be the product of a 34-week gestation, yet it weighed 6 lb. 4 oz. The infant was pale. His respirations were embarrassed, and we know that bleeding had occurred from rupture of one of the umbilical vessels.

In this particular case, we have enough evidence to determine the true etiology of the baby's difficulty. What other conditions can produce bleeding, other than rupture of a vessel due to a velamentous insertion of the cord?

1. We know that babies sometimes can bleed in placenta previa or placenta abruptio. This possibility is quite often overlooked. It is customary to think that a baby born under such circumstances is suffering from asphyxia, and to forget that the baby may lose blood, too. In about one-fifth of these cases, one can encounter the problem of fetal hemorrhage.

2. During cesarean section, the operator may incise the placenta or the umbilical blood vessels, and thus cause fetal hemorrhage.

3. Another cause is rupture of umbilical vessels as in vasa previa, velamentous insertion of the cord or overstretching of the cord.

4. There have been cases described in which the umbilical cord has been thrombosed and hemorrhage has resulted.

5. Another type of hemorrhage in this period is occult fetal bleeding into the maternal circulation. In these cases there are no gross abnormalities of the placenta. A baby is born who is pale, flaccid and apneic. Understandably, these cases are more difficult to diagnose.

Where occult hemorrhage is suspected, studies using cord blood should be done and should include hemoglobin and hematocrit determinations, blood smear, Rh typing, blood group typing, Coombs test, possibly a bilirubin and crossmatching of the baby for a blood transfusion.

I should like to emphasize the importance of ruling out hemolytic disease of the newborn due to isoimmunization. I remember an infant who at birth resembled the infant whom we are discussing today. The doctor was interested in the possibility of hemolytic disease. An Rh typing was performed and was reported as being D negative. The doctor then assumed that the baby was Rh negative, as was the mother, and therefore that there was no need to worry about the possibility of hemolytic disease. He concluded that the baby had bled into the mother, and that the immediate thing to do was to transfuse the baby. The baby had hemolytic disease. The Coombs test was strongly positive, and the negative result on the Rh typing had been due to a so-called "blocked D." Whenever one considers the possibility of hemolytic disease, he should always order a direct antiglobulin test. A blood smear is also important, as well as an examination of the baby. The infant with severe hemolytic disease will usually have an enlarged liver and spleen. The same may be true of a baby who has lost considerable blood and is in shock or in heart failure.

While these studies are being carried out, the baby should be transferred to an incubator, kept warm and given oxygen. Sometimes the situation may be so critical that the doctor may need to do something immediately and cannot afford to wait for the completion of the typing and crossmatching. A transfusion should be carried out using group O, Rh negative, low titered A and B blood. When time permits crossmatching, the baby should be given type-specific blood, and as an added safeguard against hemolytic disease should be given an indirect Coombs test, using maternal serum against the donor erythrocytes. The most rapid method for administering the blood is through a catheter in the umbilical vein.

How much blood should be given? Initially, this baby received 120 cc. of blood. He weighed about six pounds. That is quite a lot of blood—about 20

cc. per pound, or over half of his total blood volume. I think that probably the safest limit should be 10 cc. of blood per pound of body weight, so that in the beginning this infant should have received 60 cc. of blood, rather than 120 cc. Personally, what I should prefer to do, rather than to give a whole-blood transfusion to a baby as sick as this one, would be to insert a catheter in the umbilical vein, use sedimented or packed cells rather than whole blood, and do a partial exchange transfusion using about 75 cc. or at most 100 cc. of packed cells. I would measure the venous pressure, and if it were above 10-12 cm. H_2O, draw off some blood first, despite the severe anemia.

In doing the exchange transfusion, I would start out with 5 cc. amounts, and then possibly go to 10 cc. exchanges, depending upon the baby's condition. By doing this, I think I could raise the hemoglobin more effectively, and secondly, I wouldn't have to worry about the hazards of overloading the baby's circulation as much as with a blood transfusion. I would try to end with a venous pressure of 6-8 cm. H_2O.

It doesn't take very long to obtain sedimented cells. The blood has been setting in the blood bank, and the cells have sedimented. All that is necessary is to aspirate the supernatant plasma, and in just a few minutes the blood is on its way to the delivery room. Prematures tolerate cold blood poorly, and thus if time permitted, I would warm the blood in lukewarm water for five to ten minutes.

After performing the initial exchange transfusion, I would wait for the infant's condition to stabilize, and then get a hemoglobin or a hematocrit to see what had been accomplished. If the infant were still anemic, I would do another small exchange transfusion to bring the hemoglobin up to around 10 Gm.

This baby was treated with blood, and his color became pink. His cardiac rate increased to 80 per minute and later to 98, although his respirations remained embarrassed. At 8:00 p.m. an additional 20 cc. of blood was administered. The baby's heart rate increased, and his color remained good. Calcium gluconate was administered. Later, another 25 cc. of whole blood was given. The systolic blood pressure was 80 mm. Hg, and the heart rate was 100. At 11:30 p.m., the baby was somewhat pale, but was more active. His hemoglobin, however, was 14.4 Gm., which is good for a baby who had gone through everything that this infant had. But at 2:30 a.m., about 12 hrs. after birth, the child started to bleed from the penis, the nose and the rectum. What caused this new complication?

Following massive blood transfusions, bleeding may occur. We know that thrombocytopenia can occur, that the fibrinogen level may drop after massive blood transfusions and that the prothrombin level may also be affected. It is possible that the hemorrhage in this case may, in part, have been the result of the massive blood transfusion. A major factor, however, is the anoxia secondary to the initial hemorrhage and shock. Increased capillary permeability results from prolonged anoxia. I have seen anoxic infants bleed for long periods of time following routine heel puncture done in the process of obtaining blood for a hemoglobin or hematocrit. When venous blood is collected in a test tube, it doesn't clot. The plasma clotting factors also are reduced in anoxia.

Third, when a baby loses blood, as in this case, the levels of these clotting factors are reduced.

Fourth, in some prematures the level of these clotting factors may be lower than in full-term infants.

Many years ago, hemorrhagic disease of the newborn seemed to be more common. Then came the era of giving the mother vitamin K, or of giving it to the baby. Hemorrhagic disease of the newborn became less and less common. Many felt that this change was the result of vitamin K administration, but there were disbelievers who felt that vitamin K had little influence, and they stopped using it. The pros and cons are argued among physicians, and I shan't go into them. I shall merely say that the question has not been settled.

My impression is that the great reduction in the incidence of hemorrhagic disease of the newborn is more directly related to the improvements that have taken place in obstetrics and in neonatal management, than to the administration of vitamin K. For example, with better obstetrics came a reduction in the numbers of anoxic infants born following difficult and traumatic deliveries. It was not uncommon to see these severely asphyxiated infants hemorrhage around the second to fourth days of life. The prevention, or early recognition and treatment, of such conditions as severe hemolytic disease due to isoimmunization, hyperbilirubinemia due to hemolytic disease or to drugs, or immature liver function, fetal hemorrhage, sepsis, syphilis, neonatal thrombocytopenia—all accompanied by a tendency to hemorrhage—likewise brought a reduction in the incidence of hemorrhagic disease of the newborn. The influence exerted by improvements in maternal diet has not been fully evaluated.

Now we come to an important question: "How should we treat an infant who is hemorrhaging as this one was?" Are vitamin K and blood enough? First of all, we should try to understand the cause of the bleeding as best we can. It isn't difficult to obtain blood from the umbilical vein for determinations of clotting time, prothrombin time, fibrinogen level, and platelet count, and to check for fibrinolysins, anticoagulants and so forth. I am sure that if we were to do these things we would find in many cases that the clotting factors were decreased and in addition would discover a vascular defect.

This baby was given fresh whole blood. Was there anything better to do? What about the use

of blood that has been collected in heparin, rather than in ACD solution, or blood collected using siliconized material in order to preserve platelets more efficiently? Best of all, would be to have the donor present in the nursery and to draw off 20 cc. increments of blood in siliconized syringes, and then give it to the baby without using anticoagulants. Vitamin K oxide acts quickly when given intravenously, and should be given in this fashion.

In this infant, I should have resorted to the administration of steroids intravenously at the first sign of his hemorrhagic diathesis. I have no good reason for this except that in neonatal thrombocytopenia it helps even before there is a rise in the platelet count. I don't know of any studies dealing with the effect of steroids on the blood vessels.

The administration of fibrinogen has to be considered. A low fibrinogen level would call for its use.

Despite all these measures, I still might not have affected the outcome.

In conclusion, I should like to point out that when one is confronted with a premature baby who is pale, flaccid and in respiratory distress, he shouldn't think only of asphyxia and prematurity, but also should think of posthemorrhagic shock of the premature. It may occur in a placenta previa, it may occur in a placenta abruptio, or it may be due to tears or cuts of the placenta or umbilical cord, or following fetal hemorrhage into the maternal circulation. One must consider all of these possibilities.

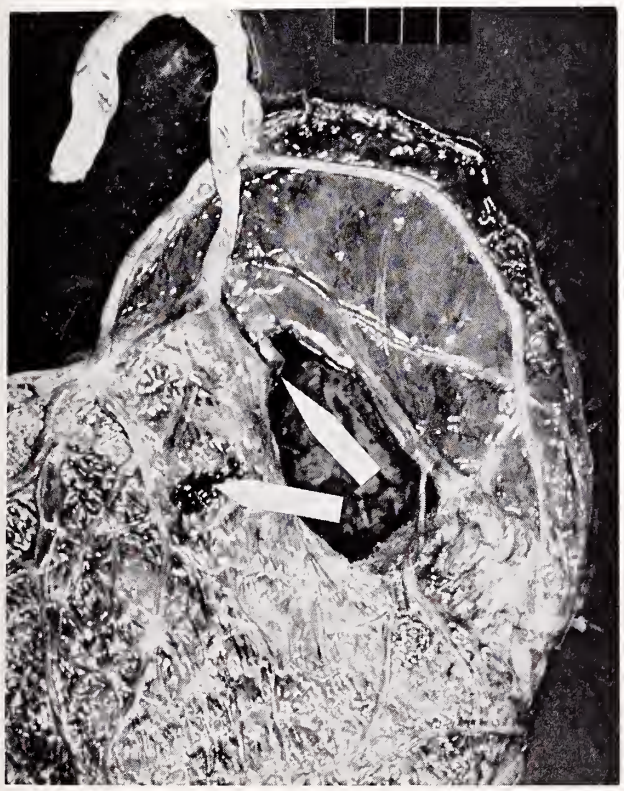

Figure 1. Placenta, showing velamentous insertion of umbilical cord. The arrows point to the ends of the ruptured vessel.

When one suspects posthemorrhagic shock, he must diagnose it and treat it promptly. I should recommend small replacement transfusions, using sedimented or packed cells. The anoxic infant who develops severe hemorrhage has a critical problem and is in great danger. He, too, must be treated quickly by means of direct transfusion, steroids, vitamin K, and in some cases fibrinogen.

Dr. Phil Meister, resident in pathology: What do you think of taking samples of cord blood from all newborns to be used later if an occasion arises and to be discarded if everything is all right after a few days?

Dr. Spevak: It might be a good idea. I don't think it impractical. *Don't throw away the placenta.* Very often, after the placenta has been thrown away, one would like to examine it, particularly in the occult-bleeding case where the baby has bled into the mother. I believe that in this hospital the placenta is routinely kept for about 24 hours. This is important, and I feel that it helps not only in this but also in several other situations.

Dr. Eugene Allende, interne: You emphasized the advisability of checking the venous pressure and then drawing off some blood even though the baby is in shock because of the blood loss. Isn't this dangerous?

Dr. Spevak: In some of these babies when one measures the venous pressure he finds it to be elevated. These babies are in shock; they are anemic; but if one measures the venous pressure by means of the catheter in the umbilical vein, he finds that the pressure is way up. The baby is pale and gasping. One cuts the cord, and the blood gushes out of the catheter. I have worried about doing this in babies who have lost blood, presumably acutely, and have done it only when the venous pressure was high enough to cause the blood to ascend the entire length of the catheter. When I finish an exchange, I try to leave the baby with a venous pressure of 6-8 cm. H_2O.

Dr. Allende: How can you be certain that the baby has bled into the maternal circulation?

Dr. Spevak: One can check for fetal hemoglobin in the mother. Sometimes the major blood groups are different, and one can do differential red-cell agglutination studies (Mollison).

There is a histochemical technic that was described by Israels in LANCET about three years ago. That method makes it possible to demonstrate fetal hemoglobin within the red cells. Fetal cells show up as "pink ghosts." One can demonstrate a few cubic centimeters of fetal blood by this technic.

Dr. O. Iglesias, interne: What has been done with regard to coagulation studies on babies who have asphyxia?

Dr. Spevak: There have been some studies. One

performed in Holland showed that anoxic infants have lower prothrombin levels. I am not completely familiar with the literature on this topic, but I am sure that there have been several studies. Most of you would agree that in anoxia one gets alteration in the functional integrity of the blood vessel. Just as any other cell in the body is affected by anoxia, so are the cells of the blood vessel. As a result of anoxia, increased vascular permeability develops.

AUTOPSY FINDINGS

Dr. Leonard E. Wallace, Pathology: The autopsy findings in this infant consisted of (1) ecchymoses over the entire body, especially on the trunk; (2) a varus deformity of the left foot; (3) bloody fluid exuding from all body orifices; (4) distention of the gut by liquid blood; and (5) massive hemorrhages in both kidneys and within the brain.

The infant weighed 3,000 Gm. A total of approximately 180 cc. of blood had been administered. This volume represented approximately 80 per cent of the infant's total blood volume. The infant was pale, and had had depressed cardiac activity at the time of delivery. The respirations had been poor. Because of extreme shock, uncrossmatched type O Rh-negative blood had been administered immediately.

The transfusion had been started prior to the time we received blood for laboratory determinations. Blood from the infant was subsequently obtained, and after two hours no clot was observed. The prothrombin time was greater than 60 seconds. The prothrombin consumption was within the normal range. No fibrinolysins were demonstrable. The fibrinogen concentration was less than 50 mg. per cent. The serum calcium was 9.6 mg. per cent.

Similar results were noted on analysis of the blood obtained shortly after death. These values, however, represented determinations on material containing the ACD solution that had been used as an anticoagulant in the bank blood. It is important, of course, to obtain blood prior to the time transfusions are started, if at all possible.

Certainly the hemorrhagic diathesis occurred secondarily to the loss of multiple factors necessary for coagulation, yet these results would suggest that the first stage of coagulation, the formation of thrombin, was intact and that there was marked depression of the fibrinogen concentration.

A careful examination of the pulmonary tissue didn't reveal the presence of amniotic contents within pulmonary vessels. One might have expected to see this material since the fetal circulation had been exposed to the amniotic fluid *in utero* at the time of rupture of the umbilical vessel. The fibrinogen depression, however, was considered to be directly related to the deficiency of this substance in bank blood.

Annual Assembly, Omaha Mid-West Clinical Society

The Twenty-ninth Annual Assembly of the Omaha Mid-West Clinical Society will be held October 30 through November 2, at the Civic Auditorium in Omaha. Alumni meetings will be held at the medical schools of the University of Nebraska and Creighton University at about the same time. Those of the University of Nebraska will be held during the preceding weekend, and those of Creighton October 30-November 2.

The guest speakers for the Clinical Society's meeting are to include A. E. Bennett, M.D., associate professor of psychiatry at the University of California; Howard P. House, M.D., clinical professor of otolaryngology at the University of Southern California; John W. Kirklin, M.D., of the Mayo Clinic Division of Surgery; Gordon McHardy, M.D., clinical professor of medicine at L.S.U.; John H. Moyer, M.D., chairman of internal medicine at Hahnemann Medical College; John W. Rebuck, M.D., hematologist at Henry Ford Hospital; Fred C. Reynolds, M.D., professor of orthopedic surgery at Washington University, St. Louis; Alexander J. Schaffer, M.D., associate professor of pediatrics at Johns Hopkins Medical School; Harry M. Spence, M.D., clinical professor of urology at Southwestern Medical School, Dallas; J. Robert Willson, M.D., head of obstetrics and gynecology at Temple University School of Medicine; and Kenneth W. Warren, M.D., a surgeon at the Lahey Clinic.

The program is co-sponsored by the Nebraska chapter of the American Academy of General Practice, among other organizations, and members will be granted hour-for-hour Category I credit for attending. The registration fee is $12 for either three or four days, or $10 for one or two days. Additional information and copies of the program can be secured from the office of the Omaha Mid-West Clinical Society, 1613 Medical Arts Building, Omaha 2.

State University of Iowa College of Medicine

Clinical Pathologic Conference

SUMMARY OF CLINICAL FINDINGS

A 44-YEAR-OLD MAN was followed at this hospital for 30 years for arthritis, but his last illness began two years before admission when he developed a "cold," sore throat and cough that lasted for two months and then disappeared while he was being treated with penicillin. Twenty months before admission, his belt became tight, and his feet and legs were swollen. His temperature remained normal. He was treated with digitalis and mercurial diuretics, with temporary diuresis and improvement. Recurrent episodes of weight gain and shortness of breath prevented him from working. Eight months prior to admission, albumin was found in his urine, but no cells. Six weeks before admission, swelling of the abdomen and legs began to increase in spite of all medication, and progressed to involve his face, hands and arms. He said his blood pressure had been only slightly above normal. A mild, non-productive cough was present.

No history of alcoholism was obtained. At age 14, swollen, painful ankle and knee joints had been observed for three or four months. This phenomenon recurred several times, but not after age 20, and there were no associated systemic symptoms. At age 23, back pain had begun, and arthritic changes had progressed to immobility of the entire spine. Pain and limited motion of hips were also present, as well as a 30-pound weight loss. The white blood cell count was 10,000/cu. mm., but the hemoglobin and sedimentation rate remained normal, and all urines were negative for albumin and cells during that early period. His blood pressure at age 23 had been 120/85 mm. Hg.

Physical examination at the time of the patient's last admission, at age 40, showed a well-developed, well-nourished white male who was alert and cooperative, but edematous to his arm pits and short of breath. There was no periorbital edema. The ocular fundi showed poorly outlined discs, decreased diameter of arteries, arteriovenous nicking, and no exudate or hemorrhage. The thyroid was not enlarged. Moist rales were heard over the entire chest. The cardiac rate varied, being either 80 or 120 per minute in runs of regular rhythm that changed suddenly. A diastolic murmur was heard in the left third interspace, in the parasternal area. A diastolic gallop was also heard. The apical sounds were distant, and a point of maximum impulse could not be palpated through the very edematous tissues. The blood pressure was reported as 155/95, 200/100 and 160/115 mm. Hg by three examiners. The total body weight was 186 lbs. at the time of admission. The abdomen was edematous and greatly distended, and there was a demonstrable fluid wave. The genitalia were also edematous. The legs were dark brown, indurated and greatly swollen with fluid.

The serum cholesterol was 347 and 466 mg. per cent on two analyses. The thymol turbidity was 9.7. The blood urea nitrogen and creatinine were 29 and 0.7 mg. per cent, respectively. The serum potassium was 4.8 and the sodium 126 mEq./L. The lupus erythematosus preparation, latex and bentonite agglutinate tests were negative on two occasions. The antistreptolysin titer was 166. Consultation in radiology resulted in the following reports: cardiomegaly; severe congestive heart failure and probably pleural effusion; normal intravenous pyelogram. Consultation in urology resulted in the conclusion: "no evidence of obstructive GU disease." Five bacterial studies of blood, two of urine and one of stool revealed a beta hemolytic Streptococcus in one blood sample, sensitive to penicillin and three other drugs; and revealed Escherichia coli, Proteus vulgaris and Pseudomonas aeruginosa in cultures of urine. Electrocardiographic tracings (five of them) showed, at various times, a normal sinus rhythm with depressed RS-T segments, a wandering pacemaker with slow nodal rhythm shifting to sinus tachycardia, an intraventricular conduction defect and, on the last tracing, a sinus rhythm with first-degree atrioventricular heart block, incomplete right bundle branch block, right ventricular hypertrophy and P-wave abnormalities. Eleven urine analyses routinely showed albumin, and 24 hr. amounts varied from 1.2 to 5.8 Gm. The specific gravities were 1.015 to 1.022 with a urine volume average of 1,320 ml.—standard deviation 246 ml. and a range up to 5,000 ml./day. There were many hyaline granular casts, occasional white blood cells and a few bacteria.

The patient was treated with low-sodium diet, digitalis, potassium and ammonium chloride, and a variety of diuretics at various times, including Mercuhydrin, aminophyllin, Aldactone, Thiomerin and chlorothiazide. He was given cortisone and related drugs. Late in his treatment, he was given vancomycin and aqueous penicillin. A renal biopsy was done, and it showed material between the basement membrane and epithelium of the glomeruli and obliteration of foot processes by electron microscopy.

His progress was one of continued edema and

gradual deterioration, from alertness to apprehension to episodes of semi-coma, decreased sensation and motor paralysis of the right leg and foot. Because he ate and drank poorly, he was given intravenous fluids. Nine days before his death, the blood urea nitrogen was 28 and the creatinine 0.9 mg. per cent, and the serum CO_2 content (exposed to air) 23.6, the chloride 89, the potassium 5.2 and the sodium 123 mEq./L. He was semi-comatose at that time. He was given 1,000 ml. of 5 per cent glucose in water intravenously on the following day, followed by 100 ml. of glucose in normal saline, and then additional 5 per cent glucose in distilled water at 5-10 drops per minute to keep the intravenous open. It was stated at that time that the patient had a "balanced salt loss."

His weight was 154 lbs. The urine output five days prior to death was 1,800, and on the following day it was 1,500 ml. Four blood protein levels ranged from 5.2 to 5.9 Gm. per cent, with albumin 0.7 to 1.4 and globulin 4.3 to 4.8 Gm. per cent. A single protein bound iodine was found to be 1.9 micrograms per cent. The last serum electrolyte determinations revealed a serum sodium of 147, potassium 3.9, CO_2 29, and chloride 89 mEq./L., and blood urea nitrogen and creatinine 22 and 9.9 mg. per cent, respectively. The blood pressure remained around 160/80 mm. Hg, except for a 24 hour period several days before death when it was 210/120 mm. Hg. The patient's temperature varied from 96 to 102°F., until the last five days when it remained below 99°F.

SUMMARY OF CLINICAL DISCUSSION

Mr. James Lichty, junior ward clerk: First, I should like to ask some questions about the patient's blood picture. No mention has been made of the hemoglobin values or red blood cell counts during his hospital stay.

Dr. Paul M. Seebohm, Internal Medicine: The hemoglobin was 13 Gm./100 ml. on admission, and 10 Gm./100 ml. several days before he died. The white count was 10,000/cu. mm. on admission, and 15,000 on the day before death.

Mr. Lichty: When confronted with this case, we were immediately aware of several possible explanations for the gentleman's difficulties. We therefore decided to pick one condition that we felt to be present in the patient and to investigate it. We felt that there was little doubt that he manifested the nephrotic syndome, so we chose to build our investigation around it.

There are four classic findings in the nephrotic syndrome all of which were demonstrated in this patient: albuminuria, hyperlipemia, peripheral edema or ascites, and hypoalbuminemia. Four of the recognized causes of this syndrome are subacute glomerulonephritis, membranous glomerulonephritis, amyloidosis involving the kidney, and lipoid nephrosis in children. We centered our attention upon two of those diseases—namely membranous glomerulonephritis and amyloidosis of the kidney. We felt that there was no good evidence to support the idea that he had had active acute glomerulonephritis that could eventually have progressed to the subacute form, and we ruled out lipoid nephrosis on the basis of the patient's age and findings which would not be typical in the electron microscopy evaluation of the renal biopsy. We couldn't completely rule out amyloidosis, but felt that it wouldn't be consistent with the electron microscope picture afforded us in the report on the renal biopsy.

In our investigation, we used the classification of Ellis regarding kidney disease, and we believe that this patient would fall under Ellis's type II nephritis. The man may have had a streptococcal infection 20 to 24 months before the onset of his symptoms, but nothing of the sort was demonstrated by culture. He was, however, treated with penicillin, and had relief of his symptoms in a period of weeks following the administration of this drug. Type I nephritis, according to Ellis, follows a known previous streptococcal infection within 7 to 21 days, and because this patient's history is incompatible with that idea, we feel that he didn't have Ellis's type I nephritis.

As I recall, the patient's admitting symptoms were swelling of his feet and legs, and tightness of his belt. Aside from these, he had few constitutional symptoms. This insidious onset of edema is one of the hallmarks of Ellis's type II nephritis. It has also been noted that patients presenting with these symptoms of insidiously developing edema very frequently perish from their disease—a fate that befell the man who is under discussion this afternoon. We feel, therefore, that this man did have Ellis's type II nephritis, that it was manifested as membranous glomerulonephritis, and that it eventually produced the nephrotic syndrome and the patient's downhill course.

I should now like to trace this man's history, as given in the protocol, and to pinpoint some of the other findings that are pertinent in his case. At age 14, the patient is known to have had swollen, painful ankle and knee joints. We were unable to determine the exact cause of these findings, but in retrospect, there are several conditions that deserve consideration. Quite possibly the patient could have had rheumatic fever. He could also have been experiencing the early manifestations of Strümpell-Marie ankylosing spondylitis, or he may have had rheumatoid arthritis. Other collagen diseases such as lupus erythematosus are also possibilities, and thus we were unable to say without question just what the true etiology was.

The protocol mentions that at age 23 the patient had the onset of immobility of his entire spine. We feel that this was probably the previously mentioned ankylosing spondylitis. It is interesting to

note that about 4 per cent of patients who have this disease also have involvement of the aorta, which manifests itself as aortic regurgitation at some later date. Although only a few individuals develop this aortic lesion, we feel that this patient had aortic disease in association with the spondylitis.

It is mentioned in the protocol that this man was followed for approximately 30 years at this hospital for arthritis. We are not told whether his disease was peripherally occurring arthritis, or whether it was confined solely to his spine. It is quite possible that he was followed over that period of years for ankylosing spondylitis.

We then note the onset of the patient's last illness. It began about two years before his admission to this hospital. At that time, he developed a "cold" and it was treated with penicillin. We can't be certain that his ailment was a streptococcal sore throat, for there is no mention that bacteriologic studies were done. The best we can say is that he may well have had a strep throat which was treated with penicillin.

We then see that he had complaints of edema, as evidenced by his swollen feet and legs. We feel that at that stage the patient was beginning to have early manifestations of the nephrotic syndome. There was no albumin in his urine at that time, but we still feel that the edema then present was due primarily to kidney disease and not to heart failure. He was treated with digitalis, with some good effects, but he had a prompt return of his retention of fluids, despite the digitalis therapy, and we feel that this was good evidence that his primary disorder involved the kidney.

At the time of the patient's final admission to this hospital, he showed signs that were definitely compatible with the nephrotic syndrome. We feel that he had cardiac failure secondary to fluid retention. His diastolic murmur may possibly be explained as an accompaniment of Strümpell-Marie disease, or he may have had aortic involvement secondary to rheumatic fever that had occurred at age 14. Of this we can't be certain. We note that during his hospital stay, one out of five blood cultures revealed a streptococcal septicemia. It is therefore possible that the murmur heard at that time was due to subacute bacterial endocarditis, if the patient had valves that had been damaged by a previous episode of rheumatic fever.

We feel that he had mild hypertension, as indicated by the three blood pressure readings. There is some discrepancy among the three levels reported, but the fact remains that he was mildly hypertensive.

It is also noteworthy that laboratory studies for lupus erythematosus were negative on two successive occasions. This finding helped us rule out lupus, although negative laboratory tests don't unequivocally prove the absence of that disease. Latex and bentonite tests for rheumatoid arthritis were also reported as negative. These tests are reputed to be positive in only 70 per cent of cases, and thus again we have only an equivocal laboraatory finding. The patient's antistreptolysin "O" titer of 166 units is certainly not diagnostic, and it is felt to be within the higher levels of normal.

It should be noted that during the patient's hospital stay, his kidney function, as indicated by his urine volume, blood urea nitrogen and creatinine, remained relatively near the normal state. This was true in spite of the fact that he was losing large amounts of albumin in his urine and had hypoalbuminemia.

It is mentioned that there were attempts made to control his cardiac decompensation and retention of fluid, and that he was also given vancomycin and aqueous penicillin. It is quite possible that those antibiotics were given him in the belief that he had subacute bacterial endocarditis.

We then come to a rather knotty problem for junior students—the electrocardiographic tracings. Because none of us could adequately interpret these findings, we consulted a member of the staff. It was his belief that in the absence of physical evidence, the EKG findings could possibly be best explained as effects of the digitalis the patient was receiving.

The remainder of the patient's hospital course was downhill. He retained more fluid and went from a state of alertness to one of semi-coma. We were at a loss for an adequate explanation of the decreased sensation and the motor paralysis in his right lower extremity. Assuming that the patient had subacute bacterial endocarditis, it is possible that there was embolization to the brain. Uremia is another possibility, but according to the laboratory values, this state didn't develop until quite late in the patient's course. His blood pressure levels indicated only mild hypertension, but a cerebral vascular accident is not beyond the realm of possibility.

In postulating the cause of this man's death, we feel that he could have died from cardiac failure, which is one of the common causes of death in patients having Ellis's type II nephritis. Or he may have died from septicemia or from an embolus to the central nervous system.

Dr. Wallace W. McCrory, Pediatrics: I'm quite willing to admit that this disease, ankylosing spondylitis, is remarkable to me, first, because of its rarity in pediatric patients. Dr. Jacqueline Noonan and Dr. Robert Gauchat have shared with me some of their thoughts on how we, as pediatricians, would have handled this patient.

I think that Mr. Lichty has done an elegant job of highpointing a number of the facets of this disease. The onset of the patient's difficulties being of concern to us, I would put my emphasis on a few different facets. When this 44-year-old man came in, he was edematous to his armpits, and his story was compatible with the fairly recent

onset of renal disease. I think that this patient had a considerable number of things wrong with him before the onset of his renal disease, but the first thing that I would interpret somewhat differently would be to lay greater stress on his major complaint in accounting for his ultimate demise. I should like to ask at this point whether the patient's complaints centered around pain in the spine and pain in the hips, so that it was ankylosing spondylitis that kept him continually coming back to this hospital.

Dr. Seebohm: Yes.

Dr. McCrory: I shan't spend time trying to differentiate the various illnesses that this man may have had at the age of 23. I think that one of the possibilities that Mr. Lichty mentioned was most likely. Let us assume that the arthritic manifestations at that time were the first signs of what later evolved as ankylosing spondylitis, and not rheumatoid arthritis. I assume now that this patient didn't have rheumatoid arthritis. It is conceivable that his illness two years before his demise here, with cough, sore throat and treatment with penicillin, was a streptococcal infection, and that this incited his terminal problem of renal disease.

The physical examination at the time of his admission showed a well-developed, well-nourished white male who was alert and cooperative, but edematous to his armpits and short of breath. There was no periorbital edema, perhaps because the patient was bedridden at that point, and was sitting.

The funduscopic findings would be compatible with changes associated with vascular disease. He did not have exudates. The eye findings were compatible with primary vascular involvement and not with nephritic retinopathy. The moist rales over the chest, I would attribute to cardiac failure, which was present in this patient.

Now, I should like to return to a comment in the first paragraph in the protocol. This patient developed recurrent swelling associated with shortness of breath. Now, unless this shortness of breath was due to elevation of the diaphragm, secondary to fluid accumulation (ascites), I should take the association of shortness of breath and edema as evidence indicating that congestive failure in this patient contributed at the beginning to the occurrence of edema and to the evolution of the nephrotic syndrome.

My interest centers on heart disease as the major problem in this patient, with the nephrotic syndrome constituting a complication during his last two years. The cardiac findings are compatible with the aortitis which, as you have heard, is seen in patients with spondylitis. The absence of any findings of mitral involvement I think may be important. We all know the difficulties of being certain that the absence of auscultatory findings in a patient with edema really means that there is no heart disease. I think it is important for us to bear in mind that there may have been a mitral involvement in this patient that wasn't evident on physical examination.

I should now like to ask whether the x-rays indicated right heart enlargement.

Dr. Carl L. Gillies, Radiology: This film is an intravenous pyelogram that was reported as normal. It demonstrates well the ankylosing spondylitis or poker spine. There was ankylosis of both hips. The left had had a cup arthroplasty, and the right had had a fascial arthroplasty.

The first film of the chest shows that the heart was enlarged and that there was pulmonary edema, but in the second and third films, taken three weeks later, the edema had cleared up, and the heart had decreased in size. The lungs show emphysema and fibrosis.

Dr. McCrory: The clearing of the signs of pulmonary failure in this patient have just been demonstrated to me by x-ray. This occurred approximately one month after the patient came in. What were the remaining findings after cardiac examination? I would assume that the patient's status was better at that time than at any other point during his terminal illness. Do you know the other findings?

Dr. Seebohm: No murmurs were heard.

Dr. McCrory: Having seen the x-rays, all of us should regard the diagnosis of ankylosing spondylitis with spine and hip involvement as fairly obvious. The information we've been given also indicates that this patient did show some response to the therapeutic measures.

At the time of his admission, we had a patient with the nephrotic syndrome and arthritis. There may also have been cardiac disease and aortitis. Whether the latter were present in this patient or not isn't yet clear, but we can't exclude the possibility.

What, then, was the cause of his nephrotic syndrome? We can be certain that he had it. There was an elevation of cholesterol and proteinuria, and there was a low level of serum albumin. I think it is of interest that his urea nitrogen and creatinine were perfectly normal, and that his urinary output during most of his hospital stay was quite adequate. I would take these findings as evidence that he wasn't having vascular disease as a result of chronic glomerulonephritis. The nephrotic syndrome was probably a complication of some primary disease in this patient, rather than the cause of heart failure, hypertension and ultimate death.

The negative lupus erythematosus preparation, latex and bentonite fixation would help exclude rheumatoid arthritis or lupus, and that exclusion would be important, for lupus in such occasions would explain both cardiac and renal involvements. I am a little surprised that there was never any mention of hematuria. At no time were there

significant numbers of red blood cells in the urine. I think that the absence of hematuria in the patient tends in some degree to limit the number of possible causes for the nephrotic syndrome. In patients with Ellis's type II nephritis—those showing chronic glomerulonephritis—the nephrotic syndrome is associated with some degree of hematuria, and thus I should have difficulty calling this glomerulonephritis on the basis of the evidence available to me. The ASO titer was 166. The reactant necessary for the laboratory measurement of ASO is gamma globulin. Patients with the nephrotic syndrome leak gamma globulin, so that the finding of a level of 166 in a patient with hypoproteinemia and proteinuria doesn't allow one to state that the patient might not have had a highly elevated titer prior to the appearance of the proteinuria. I think I agree with the student that in this instance, probably, the ASO titer is incompatible with exposure to severe streptococcal infection and doesn't indicate to us the likelihood of hypersensitivity, as would be expected if he had a very high level.

I wonder about subacute bacterial endocarditis in such a patient, superimposed upon cardiac disease associated with his arthritis. I don't feel that we have good evidence for it in view of the failure to culture alpha Streptococcus rather than beta hemolytic Streptococcus, and secondly in view of the absence of hematuria, I think it would be somewhat unlikely. Subacute bacterial endocarditis cannot be completely excluded as a possible contributing factor to heart disease in this patient, however. It could have resulted in embolism, in his central nervous system symptoms, and in his death.

The occurrence of three positive urine cultures is of interest. I should like to ask whether these occurred before or after the urology consultation. The answer would have quite a bit of weight in my thinking. As Cass and others have made clear, instrumentation is one of the best known ways of getting Escherichia, Proteus, etc. into the urine. I think that urinary tract infection in this patient, if present, was superimposed, and that he didn't have primary urinary tract infection. The finding of beta hemolytic Streptococci in blood is, I think, a little difficult to make much out of. I presume it was treated.

As I come to the electrocardiograms that gave Mr. Lichty pause, I too have some reason to hesitate. Trying to make the most of the chronology, I should assume a course of increasing cardiac difficulty. The electrocardiographic findings don't support the existence of an isolated aortitis, and do tend to be compatible with general heart disease that probably in some way gave mitral involvement, a right ventricular hypertrophy and pulmonary hypertension, so that this patient developed left heart failure. I think that the cause of his death would not have been aortitis alone, but that there is a good likelihood that he did have failing left heart function, possibly because of the existence of valvular disease.

We can now discuss treatment. This patient, I think, developed some iatrogenic complications during the course of his three months here. There was a rigorous use of diuretics. He had acidifying salts, potassium and ammonium chloride, Mercuhydrin, aminophyllin, Aldactone (the so-called adrenal aldosterone antagonist), Thiomerin and chlorothiazide. These diuretic agents were coupled with a low-sodium diet. It is well to keep in mind that this patient's weight loss from 186 lbs. to 154 lbs., if it was accomplished by causing him to have continued sodium diuresis by giving him diuretics while he was on a low-salt diet, would result in his replacing the fluid loss with water and a very low sodium intake. He could thus develop the low-salt syndrome or salt depletion in the course of successful therapy. I mention this because he did, in effect, have internal dilution, as evidenced by serum sodium levels no higher than 125 mEq./L. until just before his death. When he arrived at the hospital, with a sodium level of 125 mEq./L., he was coherent and responsive, but as he "improved" and lost weight, the serum sodium was 123 mEq./L. at one point, and he was becoming unresponsive. I think it conceivable that he was developing some of the central nervous system manifestations of intracellular dilution and salt depletion. That can lead to confusion and coma.

More importantly, it can be associated with interference in renal function. Neither of these conditions would particularly help a patient who was already in trouble because of congestive failure.

We note that nine days before death his blood urea nitrogen was 28, his creatinine 0.9, CO_2 24, his chloride 89, his potassium 5 and his sodium 123 mEq./L. He was comatose at that time. He was given 1,000 ml. of glucose in water intravenously, followed by 100 ml. of glucose in normal saline, and then 5 to 10 drops per minute of 5 per cent glucose in distilled water to keep the intravenous open. The protocol states that at that time the patient had a "balanced salt loss." Now I'm not exactly sure what that term means. A balanced salt loss may be something like an account at Mrs. Louise Geiger's Sheldon National Bank! This patient's sodium had been embezzled, and I'm not sure that it was ever replaced.

The association of semi-coma and lack of responsiveness in a patient with edema due either to the nephrotic syndrome or to congestive failure, and who has salt depletion and is undergoing diuretic therapy, would, I think, deserve some therapeutic effort to determine whether he might do any better, symptomatically, with a higher sodium. As a pediatrician considering the situation of a patient in an older age group, I should say that an effort should have been made to get

his sodium up to 133 instead of 123 mEq./L. That would have required approximately 10 Gm. of sodium. He received essentially one. My point is that there may have been a degree of sodium depletion sufficient to prevent this patient's recovering completely from the cardiac difficulty that I think was primary.

Of the other findings, I think some of the more important ones would be the results of the renal biopsy. I should assume that the pattern of events was as follows: responsiveness to medical management, improvement in edema, the clearing of pulmonary edema that has been pointed out to us, and a decrease in heart size. We would then be left with a patient with a nephrotic syndrome and a spondylitis. At that point, with an L.E. preparation negative, what type of renal disease could this patient have had? One of the most direct ways of finding out was used here—a biopsy of the kidney. The findings could influence one's therapeutic efforts. May we see them?

Mr. Dale Huff, Pathology: We received a portion of renal parenchyma that showed no significant anatomical change on light microscopy. Electron micrographs of some glomeruli of this kidney were made, but before I explain them, I should like to review the structure of a normal glomerulus. This is a schematic drawing of a glomerular capillary showing the capillary lumen, Bowman's space, and three structures between these two spaces. The first is the endothelium. Here is the endothelial nucleus and the cytoplasm surrounding the nucleus, and as one follows the cytoplasm farther and farther away from the nucleus, it becomes a thin, attenuated sheet of cytoplasm. Next is the membrane proper. The next portion is the epithelial cell. Here is a nucleus of the epithelial cell. Here are the foot processes that are attached to the external surface of the membrane. What one sees as the basement membrane in light microscopy is really composed of the peripherally attenuated sheet of the endothelium, the basement membrane proper, and the foot processes of the epithelium.

This next slide is an electron micrograph of a normal kidney. There is the capillary lumen. Here is Bowman's space. This is a nucleus of an epithelial cell with its surrounding cytoplasm that becomes formed into foot processes which attach to the external surface of the membrane. Here is an endothelial nucleus with its surrounding cytoplasm, and here is the peripherally attenuated cytoplasm.

The next slide is a picture of the patient's renal parenchyma. Here is a capillary lumen with a red blood cell in it. Here is Bowman's space. This is the thin sheet of endothelial cytoplasm on the inside of the basement membrane. The endothelium is slightly swollen in some areas. The basement membrane proper is the rather gray-appearing membrane. It is slightly thickened, and you can see that it is rather nodular on the capillary surface, and more nodular yet on the external surface. Outside the basement membrane, dense-appearing material seems to be located between the basement membrane and the epithelial cytoplasm. There are no well-formed foot processes. They seem to be smudged and obscure. These lesions, namely the thickening of the membrane with nodularity of the external surface, the accumulation of dense material between the basement membrane and the epithelial cytoplasm, and the loss of definition of the foot processes are very widespread.

Another picture of the patient's kidney shows essentially the same changes: slightly swollen endothelial cytoplasm, thickening of the basement membrane, some sort of dense material that appears to have been deposited between the membrane and the epithelial cytoplasm, and obscure foot processes.

This higher power of the last slide displays the changes more clearly. Here is the slightly swollen endothelial cytoplasm, thickened basement membrane with the nodularity of the external surface and internal surface, deposition of dense material between the basement membrane and the epithelial foot processes, and the obscure foot processes. No definite diagnosis was made at the time the biopsy was first seen. The lesion seen on these electron-micrographs are diagnostic of the nephrotic syndrome. However, the micrographs don't reveal the underlying cause of the syndrome. Many causes of the nephrotic syndrome can be ruled out—glomerulonephritis, lupus erythematosus, amyloidosis and diabetic glomerulosclerosis. The lesion is somewhat suggestive—though not diagnostic—of membranous glomerulonephritis.

The diagnosis on the basis of these electron-micrographs is the nephrotic syndrome, with the exact cause undetermined.

Dr. McCrory: It seems to me that the patient's clinical picture suggested a reasonably decent renal function, but he did have glomerular involvement that resulted in proteinuria. At his age, amyloidosis could have caused that. The nephrotic syndrome could have been an accompaniment of hypertensive vascular disease and nephrosclerosis. I should like to ask whether the Pathology Department feels that this biopsy was compatible with either of these possibilities, or should they be excluded completely?

Mr. Huff: There was nothing to indicate that.

Dr. McCrory: Now, how do we put all of this together? I think that this patient died in balance. It would appear that at the time of his death his electrolytes were within a reasonably normal range. I feel that his major problem was cardiac disease, though it isn't clear whether this re-

sulted from myocarditis associated with the same process that caused the arthritis and gave the more diverse vascular involvement, or whether there was some increased cardiac strain as a consequence of the pulmonary problems. In any case, I should list cardiac failure as the major cause of death. The nephrotic syndrome could have been a coincidental disease provoked by infection two years before death. It certainly contributed to edema; it contributed to the refractoriness of this patient; and it impeded the therapeutic efforts. But I don't see how we can attribute the death of the patient to nephrosis. I think there is still a possibility that he contracted an endocarditis in the course of his terminal hospital stay. If we had such a finding, I think it would contribute a sensible explanation for the appearance of the terminal central nervous system problems. I think I'll stop here.

Dr. Seebohm: The physicians who cared for this patient in the hospital thought he had a nephrotic syndrome, but they were somewhat bewildered as to the specific etiology. The terminal event was confusing. The general impression was that he was in electrolyte balance, but probably had an infection that couldn't be located anatomically. The possibility that he had a staphylococcal infection following a streptococcal infection was suggested on the day before he expired.

Dr. Robert L. Givler, Pathology: With reference to the patient's kidney disease, the significant finding at necropsy was bilateral renal vein thrombosis. Anasarca, ascites and pleural effusions were present, probably reflecting the nephrotic syndrome. Other findings of particular significance included extensive pulmonary fibrosis, foci of necrotizing pneumonitis, myocardial hypertrophy and a recent myocardial infarct.

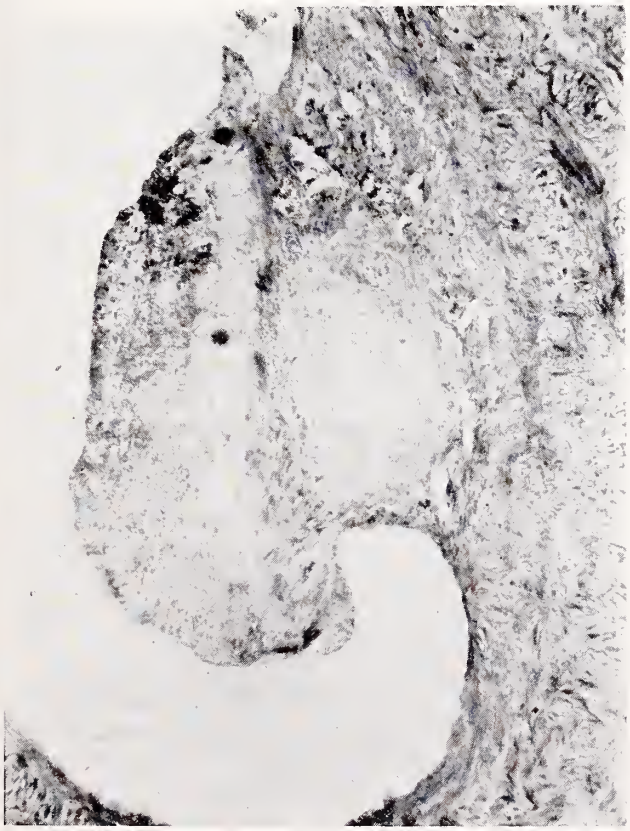

Figure 1. Renal vein branch containing mixture of old and recent thrombi. Gomori's trichrome stain.

The thrombi in the renal venous system varied in age. Both major renal veins were filled by thrombi that showed early organization in small areas. Each of these thrombi extended into the inferior vena cava for several centimeters without completely obstructing it. Larger venous branches in both renal hili contained old thrombi that were completely organized and recanalized.

Figure 1 shows a renal hilar vein partially occluded by a thrombus. This thrombus consists of both mature and organizing fibers. The microscopic features indicate that the thrombotic process began in the renal hilar veins, and that the larger thrombi filling the main renal veins represent propagations which had formed considerably more recently. The exact age of the older thrombi cannot be determined, since no further morphologic changes occur, once organization is complete. We can say only that they are of sufficient age to have been completely organized, a process which could occur in a month or less, and that their age could be consistent with the clinical duration of the patient's nephrotic syndrome. The cause of the thrombosis is not known. Sections of the renal veins showed no evidence of vasculitis.

The kidneys were enlarged, weighing 270 and 280 Gm., right and left, respectively. The renal parenchyma was not conspicuously congested. Four small infarcts were present in the left kidney. The largest measured 1.5 cm. in greatest dimension. On microscopic examination, the most striking changes were in the convoluted tubules, which showed moderate dilatation and some intraluminal proteinaceous debris. The epithelium of these tubules presented colloid droplet degeneration and cloudy swelling—non-specific degenerative changes that are generally considered to be reversible.

Figure 2 shows renal convoluted tubules in a section stained with the periodic-acid-Schiff technic. Some of the tubular epithelial cells present colloid droplet degeneration. There had been a patchy drop-out of nephron units, and they had been replaced by fibrous tissue. This change was not extensive. The majority of the renal glomerular tufts didn't show an impressive degree of alteration by light microscopy. Some were congested, and others were moderately ischemic. In sections stained with the periodic-acid-Schiff technic, the glomerular basement membranes appeared to be of normal thickness. The changes seen in the electron-micrographs of the kidney specimen taken for biopsy couldn't be seen in paraffin sections. At

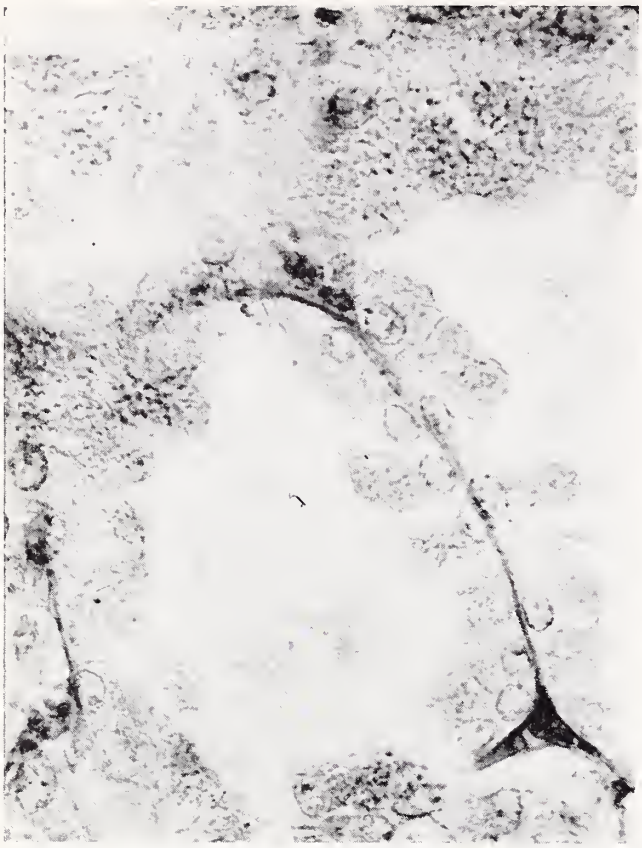

Figure 2. Colloid droplet degeneration of renal convoluted tubular epithelium. Periodic-acid-Schiff stain.

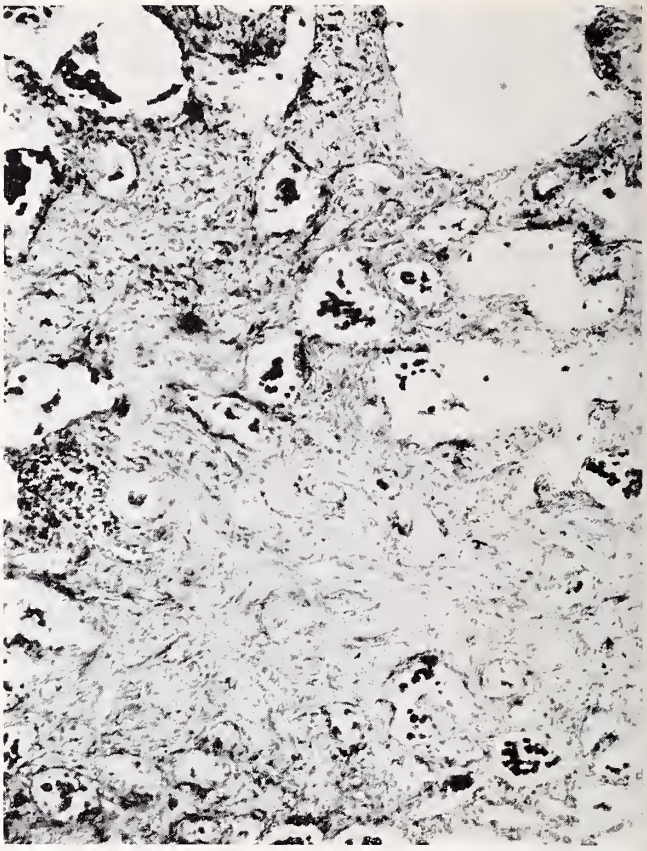

Figure 3. Pulmonary fibrosis with scattered emphysematous alveoli. Gomori's trichrome stain.

the margins of the areas of infarction, some glomerular tufts had greatly dilated capillary lumina which were filled with PAS-positive material, presumably representing intravascular thrombi. A small artery at the base of one of these infarcts contained a thrombus.

Relatively few cases of nephrotic syndrome secondary to renal vein thrombosis have been reported. Pollak and co-workers presented a comprehensive review of the subject in 1956.*

An extensive degree of pulmonary fibrosis was present. Figure 3 shows a representative area of scarring, with scattered emphysematous alveoli. The cause of this fibrosis is a matter of conjecture. Since elastic fibers tend to be more resistant to destruction than are the other components of the alveolar walls, a stain for elastic tissue is sometimes helpful in differentiating the various processes which may lead to pulmonary fibrosis. In the present case, elastic tissue stain showed the persistence of alveolar outlines within some areas of fibrosis. This pattern suggests a previous pneumonitis which healed by organization of intra-alveolar exudate, rather than by resolution. In other areas, elastic fibers were absent or irregularly tangled, indicating a greater insult, perhaps suppurative, which had been sufficient to disrupt the elastic fibers along with the other components of the alveolar walls.

Scattered foci of acute necrotizing pneumonitis were superimposed upon the chronic fibrotic process. Pseudomonas aeruginosa and Proteus vulgaris were cultivated from lung tissue at necropsy. Since the spleen cultures yielded the same organisms, we have evidence of septicemia. Microscopic examination of the brain showed several small perivascular foci of acute cerebritis. These could be attributed to septicemia.

The heart was hypertrophied; it weighed 560 Gm. Its left ventricular wall presented an area of myocardial infarction which was histologically consistent with an age of 10 to 12 days. No valvular disease was demonstrated.

Dr. Seebohm: Some explanation in regard to the electrocardiographic sequence probably should be given at this time. The day before the patient's death and several days before that, numerous electrocardiograms were taken. Other than rhythm disturbances and incomplete right bundle branch block and digitalis effects, there were no specific indications of an infarct. However, I suspect that those who interpreted these tracings thought these abnormalities could be related to an infarct. They did suggest possible metabolic disturbance as being responsible for the changes.

Now, Dr. McCrory, would you like to tell us

* Pollak, V. E., Kark, R. M., Pirani, C. L., Shafter, H. A., and Muehrcke, R. C.: Renal vein thrombosis and nephrotic syndrome. AM. J. MED., 21:496-520, (Oct.) 1956.

how frequently renal vein thrombosis explains the nephrotic syndrome?

Dr. McCrory: This instance is one more than I should have liked to observe. Although renal vein thrombosis is a rare cause, it should have been considered in the differential diagnosis. What you have heard is a perfect description of the cryptogenic course of the nephrotic syndrome occurring with renal vein thrombosis. I think it could have started two years before the patient's death. One may ask, "Why no hematuria?" In order for the nephrotic syndrome to develop with renal vein occlusion, there must be a very delicate and slow occlusion of the renal vein that will not interrupt circulation so completely that the result is infarction. The latter situation occurs more commonly. In such situations, there will be a sudden appearance of major occlusion of one or both renal veins, with hematuria, azotemia and hypertension. This patient's thrombosis was unusual because it was bilateral renal vein without venacaval thrombosis. It probably began as individual renal vein occlusion. One can sometimes get a clinical history of pain—suddenly occurring abdominal pain—and one important thing that I paid little attention to may have been the finding that the patient's legs were red and edematous. With vena caval occlusion, there would be a tendency toward much more marked edema in the lower extremity. In any case, it's fairly clear from this protocol that the condition can be completely asymptomatic and silent, for it was in this patient.

Why he should have developed this is still puzzling to me. One would have to postulate either some alteration in the major renal veins so that they would be rendered susceptible to thrombosis (and since the pathologist is shaking his head, there seems to have been no such cause), or that there was a stasis of blood flow. One can assume that there must have been some production of emboli elsewhere and that propitiously they chose to lie in both kidneys. The electron-microscopy findings on this form of the nephrotic syndrome reveal that there was a correlation between alterations in the foot processes in the basement membrane with proteinuria and the nephrotic syndrome. Thus, this finding again demonstrates the correlation we see in patients with all forms of the nephrotic syndrome. The lesion usually shows alterations in the foot processes in the basement membrane, regardless of the etiology of the nephrotic syndrome.

The clinical picture of the nephrotic syndrome seen with renal vein thrombosis has just occupied our time for about 50 minutes, and I think I need prolong the discussion no further. There may be no particular differentiating findings, and the intriguing thing is that these patients don't necessarily show the evidences of marked renal failure that one might expect. The excretory function of this patient was reasonably good. There is no effective therapy at the present time for the nephrotic syndrome that has developed because of a thrombosis of renal veins.

Dr. William E. Connor, Internal Medicine: If anticoagulant treatment had been started early in the development of this patient's thrombotic disease, wouldn't the process have been greatly altered? For example, several of the thrombi that Dr. Givler described were recent, whereas others were old and organized. Wouldn't anticoagulant drugs have inhibited the formation of those recent thrombi?

Dr. McCrory: Who would have been able to pick that up at the outset? Why was the patient thrombosing?

Dr. Seebohm: Was there any known predisposition in this patient for the thrombotic process?

Dr. Connor: An anticoagulant drug, particularly heparin, might be very helpful in patients with renal vein thrombosis, and thus it seems to me that there *is* an effective treatment, particularly if the diagnosis can be made or suspected early in the course of the disease.

Dr. McCrory: I agree with your therapeutic rationale, but how do you propose to make the diagnosis? You would have to make a renal venogram.

Dr. Seebohm: I think the problem here is how can one make this diagnosis early. I presume that everyone would agree that if such a diagnosis could be made, anticoagulants would be indicated.

Dr. Connor: One could do a radiological study of the vascular pattern of the kidneys, he could make consecutive intravenous pyelograms, or he could even undertake surgical exploration of the renal veins.

Dr. Seebohm: Would you recommend a routine venogram on each patient with nephrosis?

Dr. Connor: When a patient has a progressive nephrotic syndrome undiagnosed as to basic etiology, these additional diagnostic steps should certainly be considered. Pollak, Kark, *et al,* in the article to which Dr. Givler has referred, discussed this problem in some detail.

Dr. McCrory: Although it was rather stupid of me not to have considered the possibility of the nephrotic syndrome secondary to renal vein thrombosis, my mistake is fortuitous in that it serves to remind all of us to sharpen our diagnostic acumen when we are confronted by a patient with the nephrotic syndrome. If one can prove that the condition is secondary to renal vein thrombosis, the therapy that Dr. Connor has proposed is excellent. But the only way I can conceive of diagnosing it would be by showing interference with the blood supply. However, in view of the rarity of this etiology, I wouldn't recommend renal arteriograms as a routine diagnostic procedure in patients with the idiopathic nephrotic syndrome.

SUMMARY OF NECROPSY FINDINGS

At necropsy, the cause of the patient's nephrotic syndrome was found to have been bilateral renal vein thrombosis. The unorganized thrombi filling both renal veins extended separately several centimeters into the inferior vena cava. Some of the larger branches of the renal veins contained thrombi which were considerably older, being completely organized. Sections of the swollen, heavy kidneys showed extensive degenerative changes in the convoluted tubules. Some glomerular tufts were congested, but many were ischemic. Several areas of infarction were present in the left kidney.

The immediate cause of death may have related to an area of recent myocardial infarction having an estimated age of 10 to 12 days. The heart was hypertrophied, but the degree of coronary atherosclerosis present was not severe. Pulmonary disease included extensive fibrosis, a few foci of acute pneumonitis (Pseudomonas aeruginosa and Proteus vulgaris were cultivated from necropsy lung specimens), and several thrombi in smaller pulmonary artery branches. Sections of the brain demonstrated several microscopic foci of perivascular acute inflammation that were probably secondary to septicemia.

NECROPSY DIAGNOSES

1. Renal vein thrombosis, bilateral and chronic, with recent extension
2. Renal tubular degeneration and foci of infarction
3. Nephrotic syndrome (clinical diagnosis)
4. Myocardial infarct, recent
5. Myocardial hypertrophy
6. Pulmonary fibrosis, extensive
7. Thrombi in small pulmonary artery branches
8. Foci of acute pneumonitis
9. Foci of cerebritis
10. Rheumatoid arthritis
11. Splenic infarct
12. Fatty metamorphosis and congestion, liver.

Coming Meetings

In State

Sept. 13-14	**Pediatrics.** Medical Postgraduate Conference. S.U.I. College of Medicine, Iowa City
Sept. 23	**Eighth Annual Internal Medicine Symposium (Section of Internal Medicine, Iowa Methodist Hospital).** Des Moines Art Center, 4700 Grand Avenue
Sept. 23-24	**Anesthesiology.** Medical Postgraduate Conference. S.U.I. College of Medicine, Iowa City
Sept. 25-26	**Thirteenth Annual Meeting and Scientific Assembly, Iowa Chapter of the American Academy of General Practice.** Savery Hotel, Des Moines
Oct. 7	**Radiology.** Medical Postgraduate Conference. S.U.I. College of Medicine, Iowa City
Oct. 12	**Northeast Iowa Clinical Conference.** Masonic Temple, Waterloo
Oct. 13-14	**Arthritis and Rheumatism.** Medical Postgraduate Conference. S.U.I. College of Medicine, Iowa City
Oct. 13-14	**Urology.** Medical Postgraduate Conference. S.U.I. College of Medicine, Iowa City

Out of State

Sept. 4-7	**Tenth International Congress on Rheumatoid Diseases.** Rome, Italy
Sept. 5	**Flying Physicians Association, Inc.** Holiday Hotel, Reno, Nevada
Sept. 8-Nov. 10	**Occupational Medicine.** New York University Postgraduate Medical School, New York City
Sept. 11-16	**Vaginal Approach to Pelvic Surgery.** Cook County Graduate School of Medicine, Chicago
Sept. 11-22	**Intensive Review of Internal Medicine.** University of Southern California, Los Angeles
Sept. 13	**First Professional Kidney Symposium (National Kidney Disease Foundation).** Ambassador Hotel, Los Angeles
Sept. 14-16	**American Association for Automotive Medicine.** University of Minnesota, Minneapolis
Sept. 14-16	**New England Society of Anesthesiologists.** Wentworth by-the-Sea, Portsmouth, New Hampshire
Sept. 14-16	**Obstetrics and Gynecology.** University of California, San Francisco
Sept. 14-17	**Second International Symposium on Chemotherapy.** Naples, Italy
Sept. 15	**Lederle Symposium.** Battenfeld Memorial Auditorium, Kansas City
Sept. 15-20	**Fifteenth General Assembly of the World Medical Association.** Rio de Janeiro
Sept. 17-22	**American Fracture Association.** Shoreham Hotel, Washington, D. C.
Sept. 18-20	**Annual Fall Clinical Conference, Southwest Clinical Society.** Hotel Muehlebach, Kansas City
Sept. 18-21	**Pediatrics for Pediatricians. Center for Continuation Study.** University of Minnesota, Minneapolis
Sept. 18-22	**Internal Medicine, a Selective Review.** University of California, San Francisco
Sept. 18-23	**Surgery of Colon and Rectum.** Cook County Graduate School of Medicine, Chicago
Sept. 18-23	**General Surgery.** Cook County Graduate School of Medicine, Chicago
Sept. 18-23	**Cardiopulmonary Disease (American College of Physicians).** Ohio State University Health Center, Columbus, Ohio
Sept. 18-30	**Surgical Technic.** Cook County Graduate School of Medicine, Chicago
Sept. 18-30	**Gynecology, Office and Operative.** Cook County Graduate School of Medicine, Chicago
Sept. 23	**American Association of Hospital Consultants.** Atlantic City, New Jersey
Sept. 23-24	**American Association for Hospital Planning.** Atlantic City, New Jersey
Sept. 23-30	**Annual Otolaryngologic Assembly.** University of Illinois College of Medicine, Chicago
Sept. 24-29	**American Society of Plastic and Reconstructive Surgery.** Roosevelt Hotel, New Orleans
Sept. 25-28	**American Hospital Association's 63rd Annual Meeting.** Atlantic City Convention Hall, Atlantic City, New Jersey
Sept. 25-29	**Pathology of Diseases of Laboratory Animals.** Armed Forces Institute of Pathology. Washington, D. C.
Sept. 25-29	**Industrial Chest Diseases (American College of Chest Physicians).** Warwick Hotel, Philadelphia
Sept. 25-29	**Lower Extremities Prosthetics.** University of California at Los Angeles

Sept. 25-29 **Pulmonary Disease Seminar (University of Colorado).** Fitzsimons General Hospital, Denver

Sept. 26-29 **American Roentgen Ray Society.** Deauville Hotel, Miami Beach

Sept. 27-29 **American Association of Medical Clinics.** Barbizon-Plaza Hotel, New York City

Sept. 27, 28, 29 **Twelfth Annual Meeting of the Animal Care Panel, American Board of Laboratory Animal Medicine.** Statler Hilton Hotel, Boston

Sept. 28-30 **American Association for the Surgery of Trauma.** Drake Hotel, Chicago

Sept. 29-30 **A Clinic on Human Disabilities. University of California,** San Francisco

Sept. 29-Oct. 1 **Third International Conference of the International Medical Association for the Study of Living Conditions and Health.** St. Vincent, Aosta Valley, Italy

Sept. 30-Oct. 8 **American Society of Clinical Pathologists and College of American Pathologists.** Olympic Hotel, Seattle

Oct. 1-4 **Annual Session of the Colorado State Medical Society, combined with the Congress on Occupational Health (AMA).** Brown Palace and Shirley Savoy Hotels, Denver

Oct. 1-6 **National Recreation Congress.** Cobo Hall, Detroit

Oct. 1-7 **College of American Pathologists.** Olympic Hotel, Seattle

Oct. 2-4 **Association of Medical Illustrators.** Hotel Kahler, Rochester, Minn.

Oct. 2-4 **Obstetrics for Specialists.** Center for Continuation Study, University of Minnesota, Minneapolis

Oct. 2-5 **American Academy of Pediatrics.** Annual meeting. Palmer House, Chicago

Oct. 2-6 **Thirteenth Postgraduate Assembly in Endocrinology and Metabolism (Co-sponsored by The Endocrine Society and the National Institutes of Health).** Bethesda, Maryland

Oct. 2-6 **Latest Application of Surgical Research and New Surgical Technics. 47th Annual Clinical Congress of the American College of Surgeons.** Conrad Hilton, Chicago

Oct. 2-6 **Basic Electrocardiography.** Cook County Graduate School of Medicine, Chicago

Oct. 2-6 **Vaginal Approach to Pelvic Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 2-6 **Diagnosis and Therapy of Malignant Neoplasms of the Female Genital Tract.** New York University Medical Center, New York City

Oct. 2-13 **Clinical Uses of Radioisotopes.** Cook County Graduate School of Medicine, Chicago

Oct. 3 **American Association of Poison Control Centers.** Palmer House, Chicago

Oct. 4 **Lederle Symposium.** Broadview Hotel, Wichita

Oct. 6-7 **Western Industrial Medical Association, Western Occupational Health Conference.** Biltmore Hotel, Los Angeles

Oct. 6-7 **American Medical Writers' Association.** New York City

Oct. 6-7 **First National Congress on Medical Quackery (AMA and the Food and Drug Administration).** Washington, D. C.

Oct. 7 **Seventh Annual Meeting of the American Rhinologic Society.** (Meeting preceded by a 3-day workshop and seminar on dome preserving technics in surgery of the nasal tip, at the Illinois Masonic Hospital.) Belmont Hotel, Chicago

Oct. 7-9 **International Symposium on Bone: Clinical Application of Recent Advances.** University of California, San Francisco

Oct. 8-13 **American Academy of Ophthalmology and Otolaryngology.** Palmer House, Chicago

Oct. 9-12 **Gallbladder Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 9-12 **American Association of Medical Record Librarians.** Benjamin Franklin Hotel, Philadelphia

Oct. 9-13 **Hand Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 9-13 **The Physiologic Basis of Internal Medicine (American College of Physicians).** Duke University Medical Center, Durham, North Carolina

Oct. 9-20 **Obstetrics, General and Surgical.** Cook County Graduate School of Medicine, Chicago

Oct. 11-14 **Western Institute on Epilepsy, Thirteenth Annual Conference.** Granada Hotel and Inn San Antonio

Oct. 11-14 **Diagnostic Cardiac Auscultation.** New York University Medical Center, New York City

Oct. 12-14 **Congress of Neurological Surgeons.** Summit Hotel, New York City

Oct. 12-14 **Academy of Psychosomatic Medicine.** Emerson Hotel, Baltimore

Oct. 12-15 **Surgery of Hernia.** Cook County Graduate School of Medicine, Chicago

Oct. 13-15 **Fifth Annual Convention of the American Association of Medical Assistants.** Holiday Hotel, Reno

Oct. 14-20 **Second International Congress of Neurosurgery.** Statler Hotel, Washington, D. C.

Oct. 15-20 **Fourth International Congress of Allergology.** Hotel Commodore, New York City

Oct. 16-20 **Thoracic Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 16-20 **National Safety Council.** Conrad Hilton, Chicago

Oct. 16-27 **Internal Medicine.** Cook County Graduate School of Medicine, Chicago

Oct. 16-27 **Basic Principles in General Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 18-20 **Council on Arteriosclerosis of the American Heart Association.** Hotel Balmoral, Bal Harbour, Miami Beach

Oct. 19-20 **All That's New in Medicine. First Annual Milwaukee Medical Conference.** Milwaukee County General Hospital

Oct. 19-21 **Dermatology for Specialists.** Center for Continuation Study, University of Minnesota, Minneapolis

Oct. 20-24 **Annual Meeting and Scientific Sessions, American Heart Association.** Americana Hotel, Bal Harbour, Miami Beach

Oct. 21 **Symposium on the Problem of Obesity in Teenagers.** Stanford University

Oct. 22-25 **American College of Gastroenterology.** Hotel Cleveland, Cleveland

Oct. 22-27 **American Society of Anesthesiologists, Inc.** Statler Hilton, Los Angeles

Oct. 23-24 **American Cancer Society Annual Meeting.** Hotel Biltmore, New York City

Oct. 23-24 **Blue Shield Annual Program Conference.** Drake Hotel, Chicago

Oct. 23-27 **Advances in Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 23-27 **Gynecology, Office and Operative.** Cook County Graduate School of Medicine, Chicago

Oct. 23-27 **Clinical Cardiopulmonary Physiology (American College of Chest Physicians).** Sheraton Towers, Chicago

Oct. 23-Nov. 3 **Fractures and Traumatic Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 23-Nov. 3 **Urology.** Cook County Graduate School of Medicine, Chicago

Oct. 23-Nov. 4 **Laryngology and Bronchoesophagology.** University of Illinois College of Medicine, Chicago

Oct. 24-27 **American Dietetic Association.** Kiel Auditorium, St. Louis

Oct. 25-28 **American Association of Blood Banks.** Drake Hotel, Chicago

Oct. 25-29 **American Society of Clinical Hypnosis.** St. Louis

Oct. 26 **Lederle Symposium.** Hotel Pere Marquette, Peoria

Oct. 26-28 **Annual Course in Postgraduate Gastroenterology (American College of Gastroenterology).** Sheraton-Cleveland, Cleveland

Oct. 27-28 **Symposium on "Expanding Goals of Genetics in Psychiatry (1936-1961)."** New York State Psychiatric Institute, 722 West 168th Street, New York City

Oct. 27-29 **Association of Clinical Scientists.** Shoreham Hotel, Washington, D. C.

Oct. 28-29 **Problems Due to Infection in Medicine and Surgery.** University of California (Franklin Hospital), San Francisco

Oct. 30-Nov. 2 **Annual Assembly, Omaha Mid-West Clinical Society.** Civic Auditorium. Omaha

Oct. 30-Nov. 3 **General Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 31-Nov. 2 **Advances in Pediatric Diagnosis and Practice.** Medical College of Georgia, Augusta

Oct. 31-Nov. 2 **Interscience Conference on Antimicrobial Agents and Chemotherapy (American Society of Microbiology).** New York City

THE PHYSICIAN AND HIS ELDERLY PATIENT

In *Rabbi Ben Ezra,* Robert Browning gave to the afternoon of life a promise of better things:

"Grow old along with me!
The best is yet to be,
The last of life, for which the first was made.
Our times are in his hand
Who saith, 'A whole I planned,
Youth shows but half; trust God; see all, nor be afraid!' "

Jaques, in *As You Like It,* describing the seven ages of man, paints quite a different picture of the advancing years:

"The sixth age shifts
Into the lean and slipper'd pantaloon,
With spectacles on nose and pouch on side
His youthful hose, well saved, a world too wide
For his shrunk shank; and his big manly voice,
Turning again toward childish treble, pipes
And whistles in his sound. Last scene of all,
That ends this strange eventful history,
Is second childishness and mere oblivion
Sans teeth, sans eyes, sans taste, sans everything."

The last chapter of Ecclesiastes with great eloquence describes the declining years of man's life:

"Remember now thy Creator in the days of thy youth, while the evil days come not, nor the years draw nigh, when thou shalt say, I have no pleasure in them;

While the sun, or the light, or the moon, or the stars be not darkened, nor the clouds return after the rain;

In the day when the keepers of the house shall tremble, and the strong men shall bow themselves, and the grinders cease because they are few, and those that look out of the windows be darkened,

And the doors shall be shut in the streets, when the sound of the grinding is low, and he shall rise up at the voice of the bird, and all the daughters of musick shall be brought low;

Also when they shall be afraid of that which is high, and years shall be in the way, and the almond tree shall flourish, and the grasshopper shall be a burden, and desire shall fail; because man goeth to his long home, and the mourners go about the streets;

Or ever the silver cord be loosed, or the golden bowl be broken, or the pitcher broken at the fountain, or the wheel broken at the cistern

Then shall the dust return to the earth as it was; and the spirit shall return unto God who gave it."

Which picture fits the individual as the shadows lengthen will depend on many factors: intrinsic factors of constitution, temperament, spiritual strength, mental and physical health, and the extrinsic influences of family, friends and environment.

The physician, perhaps more than any other individual, can assist the elderly in their adjustment to the physical and emotional impact of aging, to allay fear and augment courage. It is the doctor who will hear their grievances and frustrations, who will treat the ravages of old age with tenderness. It is the physician who will make every effort to rehabilitate the handicapped and to direct their energies to creative and useful effort. It is the physician who can offer a promise of better things,

"The best is yet to be,
The last of life, for which the first was made."

THE HAZARDS OF SURGERY FOR UNSTABLE PATIENTS

F. A. Whitlock,* a British psychiatrist, recently directed attention to a causative relationship between partial gastrectomy and subsequent alcoholism, drug addiction, or psychoneurosis with a special tendency to alcoholism. Individuals who had been reasonably adjusted prior to operation became chronic alcoholics after surgery.

The patients all complained bitterly of the dumping syndrome, and all reported that alcohol relieved their symptoms. After gastrectomy the speed of absorption of alcohol is increased, resulting in the rapid relief of symptoms.

It was felt that in the patients so affected, the operation came as a particularly stressful experience to vulnerable personalities who thenceforth were unable to cope with the additional burden of postoperative symptoms.

In a subsequent issue of the BRITISH MEDICAL JOURNAL three physicians** from the General Hospital at Birmingham took issue with the psychiatrist. These men felt that the postoperative symptoms could be attributed to either a hypochromic anemia or to a macrocytic anemia, judging from their experience with 100 unselected postgastrectomy patients, and they doubted the pres-

* Whitlock, F. A.: Some psychiatric consequences of gastrectomy, BRIT. M. J. 5239:1560-1564, June 3, 1961.
** BRIT. M. J. 5243:50, July 1, 1961.

ence of a true dumping syndrome. It was their judgment that many of the post-cibal psychoneurotic symptoms could be attributed to low serum iron levels or vitamin B_{12} deficiencies.

It is unquestionably true that the psychoneurotic patient is a poor candidate for any type of surgery. It is readily conceivable that the impact of surgery and the usual postoperative symptoms following partial gastrectomy in a psychoneurotic individual can lead to chronic alcoholism. The possibility of nutritional deficiency is real, and the post-gastrectomy patient must be carefully managed.

The wise surgeon must be aware of the vulnerability of the unstable patient to the hazard of chronic alcoholism. Such individuals may be able to lead reasonably normal ways of life with ulcers, but may be converted into serious medical and social problems as a result of surgery.

THE PROBLEMS OF THE ELDERLY HAVE BEEN EXAGGERATED

Surfeited as we are by the many emotional articles, and on occasion vitriolic attacks, in the lay magazines and newspapers on the subject of the care of the aged, it is gratifying to encounter writing without bias or political implication. The following "pertinent observations" by Joseph S. Davis* are particularly timely and objective:

"The prospective enlargement of the older population (projected at 24 per cent in the 1960's) deservedly attracts attention. Yet the notion that our 'senior citizens' necessarily impose an increasingly heavy burden on the nation's economy is ill-founded. To an extent seldom realized, older people are increasingly self-supporting despite low incomes, if old age insurance benefits, pensions, self-service, mutual service, use of an owned home, and drafts on savings are all taken into account. Most of their needs are simple and small. Increasingly, they are provided with prepaid medical, surgical, and hospital coverages at a cost within their own means or those of their children or interested relatives. There are of course gaps to be filled, as the current drive for the expansion in such coverage at public expense testifies. Fuller utilization of talents and experience of oldsters, not only in unpaid activities but also in remunerative work if they want it, looms large among nationally justified objectives. Whatever net burden the elderly and aged really entail on the economy, moreover, will be easily borne as the nation as a whole grows younger and increasingly productive. . . . Whether the demographic developments in progress are wholesome or ominous for the longer future, I have not discussed. Let me merely add that Americans are accustomed to rise to challenges, and that our economic and social history has typically confounded both superoptimists and pessimists of all degrees."

* Davis, J. S.: Implications of prospective United States population growth in the 1960's. MILBANK MEMORIAL FUND QUARTERLY, 39:329-349, (Apr.) 1961. Mr. Davis is director emeritus of the Food Research Institute at Stanford University.

FACTORS INFLUENCING THE RATIO OF THE SEXES

The virile male has been prone to look upon the female of the species as inferior. Aristotle was author of the statement, "Woman may be said to be an inferior man." Nietzsche, perhaps with tongue in cheek, is quoted as saying, "Woman was God's second mistake." Yet, despite the male's feeling of superiority, it is the female who has proven to be biologically superior.

Throughout the world, the sex ratio at birth is 105-106 males to 100 females, a provision of nature to compensate for the biologic differences between males and females. In utero and in every age group throughout life more males than females die. The greater tenacity to life in the female is demonstrated by her longer life span in 42 species of vertebrates and invertebrates, including mammals. Human females are more resistant to disease, to irradiation, and to the stresses of life. At all ages the female has a lower caloric output and her heat regulating mechanism is more efficient. Her longer life span can be attributed to biologic qualities rather than to environmental influences. In the United States, during the period 1920 to 1958, the ratio of white male to white female age-adjusted death rates increased from 1.08 to 1.60.

In an intriguing review L. B. Shettles postulates* that it is not chance that determines the universal sex differential in the number of males and females at birth, but that "it may well result from the difference in number, size, behavior, longevity, and chromatin and gene content of the male- and female-producing spermatozoa."

Studies with the electron microscope have revealed that there appear to be two types of spermatozoa, one of which has a small and round head and is thought to contain the Y chromosome, and a second type which has a head that is larger and oval in shape and which is believed to contain the X chromosome. Observations which have been made on human spermatogenesis have shown that the Y is much smaller than the X chromosome, and the relative sizes and shapes of the most centrally-located chromosomes indicate that the smaller-headed spermatozoa contain the Y chromosome and the larger spermatozoa contain the X chromosome. In more than 200 individuals having normal semen specimens, there was a preponderance of the spermatozoa with smaller, rounded heads.

Shettles hypothesizes that "under favorable circumstances in the seminal fluid, the endocervical mucus, and intrauterine and intratubal secretions

* Shettles, L. B.: Conception and birth sex ratios; review. OBSTETRICS & GYNECOLOGY, 18:122-129, (July) 1961.

at the time of ovulation, smaller headed spermatazoa can migrate more rapidly and fertilize the ovum more often in the distal part of the tube." It is postulated that the alkalinity and reduced viscosity of the cervical mucus at the time of ovulation enhance spermatozoan locomotion, and the conception and birth of males. In support of this thesis, when conception was effected by artificial insemination, there was a ratio of 140 males to 100 females in some 9,000 births. Reports on animals state that mating or artificial insemination near the time of ovulation markedly increases the incidence of males born.

It is concluded that the frequency of families with a relative preponderance of one or the other sex is significantly greater than the expected frequencies on the basis of chance. It is possible that in human mating there may be an inherited tendency to produce children predominantly of one or the other sex. In support of this concept is a record of a family tree accurately traced from 1690 to 1946. There were 35 children born—33 were males and two females. Examination of the semen of the last offspring revealed that the spermatazoa had unusually small head lengths.

GASTRIC AND DUODENAL ULCERS

The management of gastric and duodenal ulcers has been a controversial subject for many years, with considerable difference of opinion between the surgeons and the gastroenterologists. Two recent concepts of the problem deserve serious consideration.

Duodenal ulcer is preferably treated conservatively and not by resection, according to Alton Ochsner,* and this represents the consensus of opinion in this country. Surgery is indicated only for the complications of hemorrhage or obstruction, or in the unusual patient who cannot be controlled medically. The New Orleans surgeon states that probably less than 0.5 per cent of persons with duodenal ulcer will require operation.

In contrast to the conservative management of duodenal ulcer, Ochsner emphatically urges operation in gastric ulcer—"It is my firm conviction," he says, "that in the treatment of gastric ulcer, with the exception of the very superficial gastric ulcer, these lesions should be resected. Moreover, I believe that the physician who treats a gastric ulcer conservatively assumes a responsibility that is seldom justified." Although pathologists, currently, are of the opinion that gastric ulcer seldom becomes malignant, Ochsner believes that chronic gastric ulcer can undergo malignant change. In support of this contention is the declining incidence of cancer of the stomach in this country, which can be attributed to the fact that more patients with gastric ulcer are being treated by resection. The removal of the premalignant lesion reduces the incidence of cancer.

It is particularly desirable to prevent cancer because of the uniformly bad results obtained in the treatment of clinically diagnosable cancer, in which 5-year survival rates range from 5 to 12 per cent. Ochsner is of the opinion that it is impossible to determine preoperatively whether the lesion is benign or malignant. It is impossible for the radiologist to rule out malignancy, and equally impossible for the gastroscopist and the surgeon—even at laparotomy with the specimen in his hand. Even the pathologist may require serial sections to determine malignancy. If one delays surgical treatment until the lesion is clinically cancer, it is too late!

The author introduces a note of caution, "Whereas a short course of conservative therapy under strict hospital control may not be too hazardous in the treatment of gastric ulcer, one must not be lulled into a false sense of security if the ulcer heals, since in the presence of gastric acidity a malignant lesion can ulcerate because of peptic digestion. In such instances therapy that would control the hyperacidity can permit healing of an ulcer even though malignant."

Brown, Cain and Dockerty,* of the Mayo Clinic, reported a study of 106 patients seen during the period 1944-1953 who had been diagnosed as having benign ulcerations but were found to have malignancies at operation. This group of patients were operated on when first seen because of the severity of their supposed gastric ulceration, or after two weeks or more of medical management. During this same period, 1,650 patients with benign gastric ulcers and 1,610 with gastric cancer were treated surgically.

According to Brown and his colleagues, some patients with chronic gastric ulcerations harbor occult cancer of the stomach. The incidence of malignant change has been disputed for many years, but estimates vary from 5 to 20 per cent. The 5-year survival rate for cancer of the stomach has reached a plateau of 10 to 15 per cent. Earlier surgical treatment will enhance chances of survival.

From their experience, the authors state, "Symptoms, age, and sex do not differentiate the benign from the malignant. The duration of symptoms and temporary healing may cloud the issue, and delay the correct diagnosis." Roentgenologic diagnosis is helpful, but is not as accurate as desired. Gastroscopy is of limited aid in differentiation of benign and malignant lesions. The decision for surgery is made by the joint efforts of the internist, the gastroenterologist, and the surgeon. After a discussion of their experience, they conclude, "These data strongly suggest that attempts to delineate the nature of the lesion by means other

* Ochsner, A.: Benign gastric ulcer. AM. SURGEON, **27**:471-472, (June) 1961.

* Brown, P. M., Cain, J. C. and Dockerty, M. B.: Clinically "benign" gastric ulcerations found to be malignant at operation. SURG., GYN. & OBST., **112**:82-88, (Jan.) 1961.

than by micro exam leave much to be desired insofar as accuracy is concerned."

The resectability rate in this series of 106 patients was 86.8 per cent. The 5-year survival rate for those having early operation was 75.0 per cent, and for those having delayed surgery the 5-year survival rate was 54.1 per cent.

According to the Mayo group, patients with gastric ulcers should be hospitalized for a period of intensive medical treatment, presently 2 weeks, if operation is not advised when the diagnosis is made. If, at the end of treatment, roentgenographic evidence of improvement is absent and if the patient's general condition otherwise permits, operation is strongly recommended. If, however, there is a marked reduction in the size of the ulcer roentgenographically, and if the symptoms have regressed, the medical treatment is continued, with specific instructions for roentgenograms of the stomach to be made after three weeks and six weeks, then every three months for a year, every six months for the next year, and every 12 months thereafter to determine, beyond all doubt, that healing has persisted. The medical treatment of a benign gastric ulcer is not a short-term affair, but must be conscientiously carried out over a minimum of three years.

Unquestionably, there will not be complete accord with the expressed views from the two surgical centers. However, the fact that differentiation of the benign gastric ulcer from the malignant is difficult, even in the most skilled hands, should prompt every physician to evaluate each ulcer patient more carefully and to maintain a high degree of suspicion in each patient with gastric ulcer. We should carefully ponder the question proposed by Dr. Ochsner: "Is the conservative treatment of this patient with gastric ulcer a responsibility that is justified?"

AUTOMOTIVE CRASH AND FIELD DEMONSTRATION CONFERENCE

The Fifth Stapp Automotive Crash and Field Demonstration Conference will be held at the University of Minnesota, in Minneapolis, on September 14-16, 1961. Col. John Paul Stapp, assistant for aerospace medicine, Advanced Studies Group, Brooks Air Force Base, Texas, will be in charge.

A number of medical and surgical specialists will participate in the Conference, the purpose of which is to determine how various structural parts of an automobile may contribute to death and injuries. The American Association for Automotive Medicine, founded in 1957, is cooperating with the sponsors.

Six study sessions and research projects are scheduled, in addition to the crash demonstrations.

Additional information can be secured from Mr. Merrill K. Cragun, associate director of the U of M Center for Continuation Study, Minneapolis 14.

1961-1962 MEDICAL POSTGRADUATE CONFERENCES AT S.U.I.

September 13-14	Pediatrics (Program on page 615 of this JOURNAL)
September 23-24	Anesthesiology (Program on page xlv of this JOURNAL)
October 7	Radiology
October 13-14	Arthritis and Rheumatism
October 13-14	Urology
November 10, December 13 and January 17	Otolaryngology and Maxillo-facial Surgery
November 17-18	Cardiac
December 1	Respiratory Diseases
December 5-6	Surgery
January 9-10	Obstetrics and Gynecology
February 13-16	Refresher Course for the General Practitioner

Detailed announcements regarding co-sponsors, speakers, topics, registration fees, etc. will be mailed to physicians approximately six weeks in advance of the particular programs. Inquiries should be addressed to John Armes Gius, M.D., director of postgraduate medical studies, Office of the Dean of the College of Medicine.

AMA OCCUPATIONAL HEALTH CONGRESS

The AMA will hold its 21st National Congress on Occupational Health at the Shirley Savoy and Brown Palace Hotels, in Denver, October 2-4 in conjunction with the annual meeting of the Colorado State Medical Society.

The Congress serves as a forum at which occupational health problems can be brought to the attention of acknowledged experts in the field, as well as an occasion for the presentation of papers. It is directed primarily toward the medical profession, but many of the sessions will be of interest to nurses, industrial hygienists, plant managers and others concerned with occupational health problems.

"Problems of Survival in Space" will be the subject of the annual banquet address to be given by Dr. James G. Gaume, chief of the Space Biotechnology Program of the Martin Company, in Denver. Other subjects to be discussed during the Congress include diagnosis of occupational illness by the general practitioner, relationship between the plant physician and the family doctor, workmen's compensation, the role of the occupational nurse, and the efficient utilization of the worker.

Copies of the programs both of the Colorado Medical Society's annual meeting and of the AMA Congress on Occupational Health can be secured from Mr. Harvey T. Sethman, 835 Republic Building, Denver.

President's Page

Though Dr. Homer E. Wichern, chairman of the IMS Legislative Committee, didn't have an opportunity to testify at the hearings of the House Ways and Means Committee relative to the King-Anderson Bill and similar proposals, late in July, his statement was accepted for publication with the transcript of oral testimony.

Dr. Wichern's statement will also appear in the October issue of the JOURNAL OF THE IOWA MEDICAL SOCIETY, and every Iowa doctor should read it.

It now seems unlikely that Congress will act during the remainder of this year upon any of the schemes for providing medical care to recipients of Social Security benefits. Yet, none of us can afford the mistake of thinking that the issue is dead. On the contrary, in anticipation of a crisis late next winter or next spring, we must redouble our efforts to win our fellow citizens and our lawmakers to our point of view.

President

IN MEMORIAM

Ransom D. Bernard, M.D.

August 8, 1882—July 7, 1961

In the history of the Iowa State Medical Society there are a few men that stand out on the horizon as mountain peaks for their service, leadership and devotion to the cause of organized medicine.

Dr. R. D. Bernard, or Sam as he was known to us all, was certainly one of these men. It was a great privilege for me to have been associated very closely with him through many of those years of his active life, and I treasure very much the memories of that relationship. His energy, enthusiasm, judgment and leadership were qualities given to but few men.

He was born in Elkader on August 8, 1882, and he passed away at his home in Ames on July 7, 1961, following a heart attack. His last year was saddened by the death of his only child, Mrs. Bernice Black, of Ames. He is survived by two grandchildren and by his son-in-law, Professor H. M. Black, of Iowa State University, in Ames.

Following his preparatory work in Elkader, he graduated from the University of Wisconsin in 1904. He then entered Rush Medical School and interned at Presbyterian Hospital in Chicago.

Dr. Bernard

In 1909 he entered general practice at Clarion, Iowa, where he continued for forty-odd years, interrupted by World War I, for which he volunteered in 1917. He served two years in France. He then returned to Clarion and was the first commander of the American Legion Post there.

At the time when President Truman tried to put over his socialized medicine program, the State Society was in a lethargic condition. Many offices and committees were non-functioning. It was a small office with little help. The situation was serious, and the Trustees prevailed upon Dr. Bernard to accept the post of general manager—a responsibility which he held until 1954, when he moved to Ames.

His nine years on the State Society's Legislative Committee, his experience as delegate to the AMA, and as a member of the Blue Cross-Blue Shield Boards, and the presidency of State Society in 1946 well qualified him for the reorganization that was needed in the early fifties in providing leadership for the reactivated Society.

Sometimes his somewhat abrupt manner may have startled you, but as you sat down with him and listened to his reasons, it was apparent that he had thought the problem through. His broad acquaintance and judgment of men did so much for medical public relations during those critical years. The Society tried to recognize Dr. Bernard's services in 1954, by giving him its Distinguished Service Award.

During his final year in Des Moines, his beloved wife became almost an invalid. To many of us their devotion to each other and his tender care of her emphasized the type of man he was.

Yes—he lived a positive life. His character, energy and devotion to the profession he loved laid the foundation for much of our invigorated State Society of today.

Yes—truly we are indebted to you, Sam.

—BEN T. WHITAKER, M.D.

FIRST NATIONAL CONGRESS ON MEDICAL QUACKERY

The First National Congress on Medical Quackery, which will be held in Washington, D. C., October 6-7, was conceived by the AMA's Department of Investigation. Recognizing that medical quacks and charlatans thrive in the dark shadows of public ignorance, the Department felt the meeting would be an effective way of throwing the spotlight on this problem. The Food and Drug Administration will be a joint sponsor, for it was quick to recognize the possibilities of the venture.

State and county medical societies were urged last June by the AMA House of Delegates to send representatives to this meeting, for it will be a springboard for a vigorous, multifaceted attack on medical quackery.

IN MEMORIAM

Walter Lawrence Bierring, M.D., M.A.C.P., M.R.C.P., Edin.

July 15, 1868—June 24, 1961

Late in the afternoon on Saturday, June 24, Doctor Walter Lawrence Bierring, president of the Iowa State Medical Society in 1908, died at the age of 92. Only a few days before his death he had expressed the hope that he might live to celebrate his 93rd birthday on July 15. But this was not to be. He had been confined to Iowa Methodist Hospital since February, initially with a disabling skin disorder—dermatitis herpetiformis—and subsequently with a failing circulatory system. Until the day of his death his mental faculties remained as keen and alert as ever, and until he entered the hospital in February, he had been full-time on his job as director of the State Health Department's Division of Gerontology, Heart and Chronic Diseases. Thus came to an end the remarkable career of this nationally and internationally known physician, friend to thousands and beloved by all who knew him. His many years of devoted service to his fellow-man, his wide interests and accomplishments in nearly all fields of his chosen profession, and the many honors bestowed upon him both at home and abroad, constitute a life that was lived to the fullest—a life of achievement and of service. While we may sorrow that the inevitable had to come, yet we can only feel gratitude that such a great personality lived among us for so many years. His memory will live long in our hearts and minds for his kindliness, his charm of manner, his quick and animated interest in the problems of others and above all for his distinguished service to humanity.

Dr. Bierring's entire life was spent in Iowa. He was born in Davenport on July 15, 1868. In the fall of 1889 when he was 21 years old, he enrolled in the School of Medicine at the State University of Iowa. His exceptional qualities were apparent during his undergraduate days, for during his senior year he was invited by the dean to undertake a two year postgraduate tour of European medical centers, and upon its completion to return as chairman and professor of the projected Department of Pathology and Bacteriology. In Europe he studied at the University of Vienna, the University of Heidelberg and at the Pasteur Institute in Paris. His experiences during his several trips abroad and his contacts with many of the famous European professors of that era were a source of great satisfaction to him throughout his life. In 1894, on his second visit to Europe, he spent six months at the Pasteur Institute in Paris studying under Pasteur and his associates Roux, Metchnikoff and Calmette. It was Roux who taught him how diphtheria antitoxin was made, and upon his return to Iowa he made the first diphtheria antitoxin ever made in the U. S. west of New York City. His experience at the Pasteur Institute was undoubtedly the highlight of his European postgraduate training years.

Dr. Bierring's capacity for making friends was enormous. He knew and was known by thousands in America, and he also had many friends in other countries. He knew Osler, Widal, Welch, Sir Clifford Albutt, regius professor at Cambridge, Sir Humphrey Roleston of the Royal College of Physicians, Sir Holbert Waring of the Royal College of Surgeons, Sir Normal Walker, a dermatologist in Edinburgh and Sir Donald Mac Alester, chancellor of the University of Glasgow and president of the General Medical Council of Great Britain. He traveled extensively in spite of the handicap of an artificial leg that had been made necessary by an amputation for a malignant tumor when he was a young man. In 1958, when he was 90 years old, he traveled an estimated 45,000 miles. His most spectacular trip in that year was to Beirut, Lebanon, for the purpose of installing a chapter of Alpha Omega Alpha at the University of Beirut (he was president of A.O.A. from 1924 to 1960 and installed over 50 new chapters in American universities in this country and abroad) and to deliver the opening address at the Middle East Medical Assembly. The title of his address was "Billroth, Pasteur, Osler—a Rich Heritage." Two days after his arrival in Beirut, rioting against the government began, with night bombing. His task completed in spite of difficulties and sleepless

Dr. Bierring is shown talking with Dr. Dwight H. Murray, of Napa, California, president of the AMA, at Chicago in June, 1956, on the occasion of his receiving an AMA Distinguished Service Award.

nights, he left a few days later by plane at nine in the morning. That afternoon the airport was bombed and closed to all traffic. This trip, which he made alone at the age of 90, attests the extraordinary physical stamina with which he was endowed.

Dr. Bierring's professional career was actually four careers in one. From 1893 to 1913 he was an academician. For the first 10 years he served as professor of pathology and bacteriology at the State University of Iowa. He was then appointed to the chair of theory and practice of medicine, a post he held for seven years. In 1910 he moved to Des Moines where he became professor and head of the Department of Internal Medicine at the Drake University Medical School. When the latter merged with the College of Medicine at the State University of Iowa, in 1913, he began his second career in private practice as a consultant in internal medicine. He served in this capacity for another 20-year period—from 1913 to 1933. His third career was as commissioner of the State Department of Health of Iowa—a post he accepted in 1933 at the age of 65, when he might have well been thinking of the comforts of retirement. He was commissioner of health from 1933 to 1953, still another 20-year period. But he was not yet ready to retire, and in 1953 he began his fourth career as director of the State Health Department's Division of Gerontology, Heart and Chronic Diseases.

Throughout his lifetime Dr. Bierring contributed generously of his time and talents to what might be called extracurricular activities. He served as president of the Johnson County Medical Society in 1902, of the Iowa State Medical Society in 1908 and of the Polk County Medical Society in 1911. He became a member of the American Medical Association in 1895 and was a member of its House of Delegates for some 18 years. In 1933 he was elected to the presidency of the American Medical Association, and in 1956 was awarded the American Medical Association's Distinguished Service Medal.

He was greatly interested in all phases of medical education, licensure and technics of examination. He was secretary-treasurer of the Federation of State Boards of the United States and editor of the FEDERATION BULLETIN from the time of its origin in 1915. He assisted in the organization of the National Board of Medical Examiners in 1915 and 1916, and served as its president from 1928 to 1930. In 1919 he was a member of a commission appointed by the Secretary of War, Newton D. Baker, to visit Scotland, England and France to study methods of examination in those countries. In 1922 he was made an honorary member of the Royal College of Physicians of Edinburgh in recognition of his distinguished services. He was the first chairman of the Board of Internal Medicine organized in 1936, and was issued Certificate No. 1. He also was issued the first certificate of the American Board of Preventive Medicine and Public Health established in 1947, and was its chairman for nine consecutive terms. He was instrumental in the establishment of a Sub-Board of Aviation Medicine in 1953 and of Occupation Medicine as a Sub-Board in 1955. Whether at the local, state or national level, Dr. Bierring's wise counsel, enthusiasm, willingness and ability made him a much sought after member of a wide variety of medical organizations. He held membership in some 30 of such organizations.

He was author of a number of scientific articles which have been published in medical journals. A selected "Bierring Bibliography" published in the Walter L. Bierring Festschrift issue of the JOURNAL OF THE IOWA STATE MEDICAL SOCIETY in August, 1957, lists 194 separate titles. He was also the author of several books. He gave innumerable lectures in all parts of this country and abroad. One of the most outstanding of these was the Alphonse M. Schwitalla Lecture on "Medical Echoes" given at St. Louis University School of Medicine on March 24, 1950, and published in the April issue of PHAROS in that year.

Dr. Bierring was married to Miss Sadie Byrnes, daughter of a Walcott, Iowa, physician, on April 14, 1896. Mrs. Bierring died in 1943. Surviving are two daughters, Mrs. Florence Hutchinson and Mrs. Elza B. Radoff, a grandchild and twin great-grandchildren.

Thus briefly may be recorded, in this memorial tribute, something of the accomplishments of this great man of medicine. Iowa is proud to claim him as a native son. And certainly history will acclaim him as one of the nation's outstanding physicians of all time.

—LEE FORREST HILL, M.D.

RESULTS OF M.D. PHYSICALS AT AMA

Results of the physical examinations given 1,900 physicians during the annual meeting of the AMA in New York City last June were announced on August 4. Electrocardiograms revealed heart abnormalities in 17.7 per cent of 1,945 physicians, according to Dr. Charles E. McArthur, Olympia, Washington, chairman of the committee in charge of the project.

Dr. McArthur said he was impressed with the consistency of the data collected during the seven years during which M.D. physicals have been given at AMA annual meetings. Despite the fact that each year there is a different group of examinees and different consultants, the normal electrocardiograms have approximated 80 per cent each year.

Chest x-rays of the 1,900 physicians showed: suspected tuberculosis in 5.3 per cent; other lung abnormalities in 6.1 per cent; cardiovascular abnormalities in 6.7 per cent; and other pathologies in 6.7 per cent.

THE JOURNAL *Book Shelf*

BOOKS RECEIVED

PREVENTIVE MEDICINE IN WORLD WAR II, VOL. V., by the Medical Department of the U. S. Army. (Washington, D. C., Government Printing Office, 1960. $5.75).

THORACIC DISEASES, by *Eli H. Rubin*, M.D., and *Morris Rubin*, M.D. (Philadelphia, W. B. Saunders Company, 1961. $25.00).

MAYO CLINIC DIET MANUAL, THIRD EDITION, ed. by the Committee on Dietetics of the Mayo Clinic. (Philadelphia, W. B. Saunders Company, 1961. $5.50).

THE NATURE OF SLEEP, A CIBA FOUNDATION SYMPOSIUM, ed. by *G. E. W. Wolstenholme*, O.B.E., M.B., B.Ch., and *Cecilia M. O'Connor*, M.A. (Boston, Little, Brown and Company, 1961. $10.00).

PATHOLOGIC PHYSIOLOGY, THIRD EDITION, ed. by *William A. Sodeman*, M.D. (Philadelphia, W. B. Saunders Company, 1961. $15.00).

WHAT TEENAGERS WANT TO KNOW, by *Florence Levinsohn*, in consultation with *G. Lombard Kelly*, M.D. (Chicago, Budlong Press Co., 1961. $1.50).

PROGRESS IN NEUROLOGY AND PSYCHIATRY, AN ANNUAL REVIEW, VOL. XVI, ed. by *E. A. Spiegel*, M.D. (New York, Grune & Stratton, 1961. $12.75).

CURRENT PSYCHIATRIC THERAPIES, VOL. I, ed. by *Jules H. Masserman*, M.D. New York, Grune & Stratton, 1961. $7.50).

RELIEF OF SYMPTOMS, SECOND EDITION, by *Walter Modell*, M.D. (St. Louis, C. V. Mosby Company, 1961. $11.50).

SIMPLIFIED DIET MANUAL, SECOND EDITION, prepared by the Nutrition Service of the Iowa State Department of Health, in cooperation with the Iowa Dietetic Association. (Ames, Iowa State University Press, 1961. $2.50).

HEALTH EDUCATION, FIFTH EDITION, ed. by *Bernice R. Moss*, Ed.D., *Warren H. Southworth*, Dr. P.H., and *John Lester Reichart*, M.D. (Washington, D. C., National Education Association, 1961. $5.00).

HYPERTENSION, ed. by *Milton Mendlowitz*, M.D. (New York, Grune & Stratton, 1961. $6.50).

QUINONES IN ELECTRON TRANSPORT, A CIBA FOUNDATION SYMPOSIUM, ed. by *G. E. W. Wolstenholme*, O.B.E., M.B., B.Ch., and *Cecilia M. O'Connor*, M.A. (Boston, Little, Brown and Company, 1961. $11.00).

A TRAVELER'S GUIDE TO GOOD HEALTH, by *Colter Rule*, M.D. (New York, Doubleday, Inc., 1961. $0.95).

PROGRESS IN MEDICAL GENETICS, VOL. I, ed. by *Arthur G. Steinberg*, Ph.D. (New York, Grune & Stratton, Inc., 1961. $9.75).

MEMOIRS OF A MEDICO, by *E. Martinez Alonso*, M.D. (New York, Doubleday, Inc., 1961. $4.50).

RECENT ADVANCES IN BIOLOGICAL PSYCHIATRY, VOL. III, ed by *Joseph Wortis*, M.D. (New York, Grune & Stratton, Inc., 1961. $9.75).

YEAR BOOK OF ENDOCRINOLOGY (1960-1961 YEAR BOOK SERIES), ed. by *Gilbert S. Gordan*, M.D. (Chicago, The Year Book Publishers, Inc., 1961. $8.00).

BOOK REVIEWS

A GUIDE TO ANTIBIOTIC THERAPY, by *Henry Welch*, Ph.D. (New York, Medical Encyclopedia, Inc., 1959. $3.00).

This book is an encyclopedic-type of reference work consisting of 69 pages published on May 14, 1959. It lists in alphabetical order 31 different antibiotics, some of which are only for topical use. The author states that there were, at that time, 400 different preparations of these antibiotics. The susceptibility to each antibiotic is listed for all of the commonly pathogenic bacteria, Rickettsiae, some protozoa, fungi, and two viruses. For each organism, the lowest inhibitory concentration, the highest inhibitory concentration obtainable as reported in the literature, and the concentration of antibiotic to which at least half of the strains are susceptible are listed.

This work entailed an enormous amount of research, and it is organized in a very concise and logical fashion. The description of the antibiotic, the chief indications for its use, and the side effects, as well as the usual concentrations obtained in serum and urine, are given.

This work is not apt to be of much use to the clinician, for the clinician ordinarily is faced with a certain type of infection, and the book is indexed by antibiotic rather than by clinical infection. Furthermore, the method of selecting the preferred antibiotic involves first a cumbersome comparison of serum and urinary concentrations of the various antibiotics that are obtainable with ordinary dosages, and then correlating this with the organism's susceptibility to each of the general antibiotics to which it might be responsive.

The book might be of some interest to one who deals exclusively in the infectious diseases or to a bacteriologic laboratory.—*Charles H. Gutenkauf*, M.D.

VIRUS MENINGO-ENCEPHALITIS (CIBA FOUNDATION STUDY GROUP No. 7), ed. by *G. E. W. Wolstenholme*, O.B.E., M.B., B.Ch., and *Margaret Cameron*. (Boston, Little, Brown and Company, 1961. $2.50).

This symposium on virus meningo-encephalitis reports the results of the Ciba Foundation Study Group No. 7 meeting in London on September 27, 1960. Virologists, neuro-pathologists, and clinicians reported upon their experiences in cases of aseptic meningitis and associated virus studies.

There is included a discussion of the enterovirus group, including poliomyelitis, Coxsackie, and ECHO. Arbo-virus encephalitis, tick-borne encephalitis, adenoviruses, lymphocytic choriomeningitis (L. C. M.) virus, and mumps encephalitis are also studied.

It appears that many of us find it difficult to keep abreast of current virological reports. This small volume (120 pages) is a painless way of "catching up" —or at least up to 10 months ago—upon knowledge of the viruses causing aspectic meningitis. The information is pertinent, timely, not bogged down with detail,

and well worth the short time and effort required to read this book.—*John T. Bakody,* M.D.

CLINICAL DIAGNOSIS BY LABORATORY EXAMINATIONS, THIRD EDITION, by *John A. Kolmer,* M.D. (New York, Appleton-Century-Crofts, Inc., 1961. $10.00).

The previous edition of this book, copyrighted in 1943, was in two parts. Part I was entitled "The Clinical Interpretation of Laboratory Examinations," and the second, "The Practical Application of Laboratory Examinations in Clinical Diagnosis."

The current text has been rewritten and expertly condensed to approximately one-half the bulk of its predecessor. It follows the general format of the previous Part I and contains 142 diagnostic tables and summaries.

The author is eminently familiar with both the "art" and the "science" of medicine, and describes the necessary balance between the two for intelligent patient care. Accordingly, the science of laboratory medicine is placed in its proper perspective. The text is not one of technic but one of clinical interpretation of the changes seen in the ever-increasing numbers of laboratory procedures.

Much valuable information related to laboratory medicine is readily available to any physician by having access to this text. The book is recommended especially to pathologists, pathology residents, and hospital libraries.—*David Baridon, Jr.,* M.D.

SHORT COURSE IN PEDIATRICS AT IOWA CITY

Rheumatic fever will be the subject of a postgraduate short course in pediatrics that is to be held at the Medical Amphitheatre of the S.U.I. College of Medicine, September 13-14, under the sponsorship of the Iowa Pediatric Society, the Division of Maternal and Child Health of the State Department of Health, and the S.U.I. Department of Pediatrics.

Wednesday, September 13

9:15 a.m. "Rheumatic Fever—Pathogenesis"—Lewis W. Wannamaker, M.D., professor of pediatrics, University of Minnesota, and career investigator, American Heart Association

10:15 "Rheumatic Arthritis, Differential Diagnosis"—Robert D. Gauchat, M.D.

11:00 "Rheumatic Carditis"—Alvan R. Feinstein, M.D., assistant professor of medicine, New York University, and director, Irvington House, Irvington-on-Hudson, New York.

11:45 QUESTION AND ANSWER PERIOD

12:15 p.m. LUNCHEON MEETING OF MEMBERS OF IOWA ACADEMY OF PEDIATRICS

12:30 LUNCH, DOCTORS' DINING ROOM

2:00 "Chorea"—John C. MacQueen, M.D.

2:30 "Treatment of Acute Rheumatic Fever"—Albert Dorfman, M.D., professor of pediatrics, University of Chicago, and director, La Rabida Sanitarium

3:30 CASE PRESENTATIONS AND DISCUSSIONS OF PROBLEM PATIENTS WITH RHEUMATIC FEVER—Jacqueline Noonan, M.D., and guest speakers

5:00 BUSINESS MEETING, IOWA PEDIATRIC SOCIETY

6:30 SOCIAL HOUR AND DINNER, UNIVERSITY ATHLETIC CLUB

Thursday, September 14

9:00 a.m. PANEL DISCUSSION: Prophylaxis of Rheumatic Fever—staff and guest speakers

10:30 PANEL DISCUSSION: Long-Term Management of Patients With Rheumatic Heart Disease —staff and guest speakers

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NORTHEAST IOWA CLINICAL CONFERENCE

Masonic Temple, Waterloo
October 12, 1961

8:00-9.30 a.m. Registration, visits to exhibits and "second" breakfast of coffee and doughnuts

9:30 "Clinical Aspects of Recent Cancer Research"—Robert C. Hickey, M.D., professor of surgery, S.U.I.

10:10 "Behavior Problems in Children"—John G. Young, M.D., professor of pediatrics, Southwestern Medical School, and chief of staff, Texas Children's Hospital, Dallas

11:10 "Orthopedic Problems of Lower Extremities in Children"—William D. Fischer, professor of orthopedic surgery, Northwestern University.

12:00 noon LUNCHEON, Elks Club

1:30 p.m. Subject to be Announced—S. J. Behrman, M.D., associate professor of obstetrics and gynecology, University of Michigan

2:10 "Hypocholesterol Medication"—Ivan D. Frantz, professor of internal medicine, University of Minnesota

3:10 "Management of Infections in Urinary Tract"—William L. Valk, professor of urology and chief of urologic surgery, University of Kansas

3:50-5:00 ROUND TABLE DISCUSSIONS:
"Congenital Dislocation of the Hip"—Dr. Fischer
"Behavior Problems of Adolescents and Their Parents"—Dr. Young
"Surgical Approach to Carcinoma"—Dr. Hickey
Subject to be Announced—Dr. Behrman
"Management of Cardiac Condition"—Dr. Frantz
"Tumors of the Genital and Urinary Tract" —Dr. Valk

6:00 COCKTAIL HOUR, Elks Club

7:00-12:00 DINNER AND DANCE, Elks Club

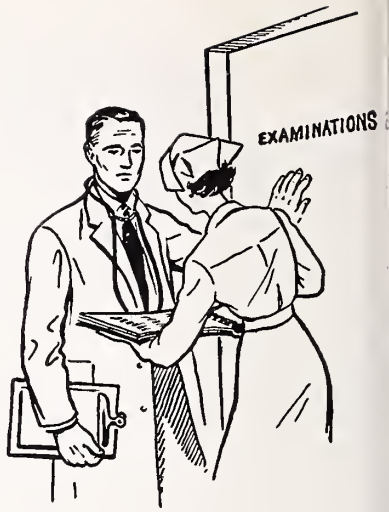
LADIES' PROGRAM

Although doctors' wives are welcome to hear any or all of the speakers, they will be especially interested in Dr. Young's subjects.

8:00-10:00 a.m. Coffee

1:00 p.m. Luncheon and Style Show

THE DOCTOR'S BUSINESS



Partnership Life Insurance

HOWARD D. BAKER
WATERLOO

Frequently the question arises in medical partnerships as to whether the prospective purchase of a partner's interest, following his death, should be funded through the purchase of life insurance. Money can be made available for this purpose through a system of cross-insurance. Each policy on a partner's life is owned by all of the other partners, and they are also the beneficiaries of that policy. For example, "A" and "B" own a policy on "C," "A" and "C" own a policy an "B," and "B" and "C" own a policy on "A." Because of the partners' different ages and insurabilities, the insurance premiums usually are totaled, and equal shares are charged to the partners. These premiums represent personal, non-deductible expense to the individual participants.

When one of the partners dies, this insurance is not a part of his estate, for the policy on his life is owned by the other partners. Moreover, the proceeds are tax-free to his former associates.

ADVANTAGES

1. Such a plan provides immediate cash for the liquidation of the decedent's capital interest in the partnership.

2. It eliminates all risk that a partner's estate may not get its money from the surviving partners.

3. It eliminates any financial burden on the surviving partners, especially when two members have chanced to die in quick succession. Without such insurance, it often is necessary to liquidate the partnership in order to pay off the interest of the deceased member or members.

DISADVANTAGES

The major disadvantages of partnership insurance are the following:

1. Indirectly, each partner finances the liquidation of *his own* capital account by paying his *pro rata* share of the cost of all policies. To the extent that his share of the premiums is disproportionately large, his payments benefit his partners, rather than his own family.

2. Advanced age and substandard insurability often make the cost of such an insurance program extremely high.

3. For partners who are financially independent, such funding may represent an unnecessary expenditure.

HOW SHOULD THE QUESTION BE DECIDED?

What, then, is the best approach to the question of whether or not a partnership should purchase life insurance on its members?

1. A determination should be made of the capital investment in the partnership. In many manfacturing and retailing firms, the capital investment represents virtually all the assets of the partners. This generally is not true of professional partnerships.

2. The age, insurability and financial status of each partner should be reviewed. This study will reveal the cost of an insurance plan and will help to determine the need for such an arrangement.

3. The practical alternatives to an insurance plan should be studied. These include such things as (a) borrowing money by mortgaging the partnership's building, equipment, etc., (b) paying the heirs from accounts receivable as they are collected, and (c) making a deceased partner's capital interest payable to his heirs in installments.

For example, the "A, B and C" partnership owns an office building valued at $54,000 and has accounts receivable of $39,000. The per-man cost of liquidation would be $31,000 in case of death. Dr. A is 57 years of age and is independently wealthy. Insurance on his life would cost 20 per cent more than the standard rate for men of his age. Dr. B is 41 and could easily borrow funds to purchase his share of a partner's interest. Dr. C is 29 and has no resources whatsoever. His maximum credit would be $5,000.

Here is the solution that they should choose. Accounts receivable can be made self-liquidating. In case of a death, they can be frozen, with each

Mr. Baker is a partner in Professional Management Midwest, and manager of its Retirement Planning Department. He majored in accounting and business administration at S.U.I., and was an agent of the U. S. Bureau of Internal Revenue for 3½ years before forming his present association in 1953.

partner to be paid his share as they are collected. A clause can be put into their partnership agreement providing that surviving partners have the option of paying off a deceased partner's interest either in full or over a period of five years, in payments of no less than $300 per month and with interest of 5 per cent on the decreasing balance. There is no reason to suppose that the heirs of a deceased member might fail to get their money since, in addition, each surviving doctor has his own credit and resources, and together the surviving partners will have a mortgagable building on which between $25,000 and $30,000 could be obtained at 5½ per cent interest.

In this case, we feel that such a solution would be much more workable than an extremely high-cost insurance plan.

In summary, we feel that large life insurance programs are usually unnecessary for professional partnerships, and that the doctors with relatively slender resources ought rather to provide insurance protection for their own families. However, this is only *generally* true, and there are individual exceptions. Therefore, an intensive study of the problem should be made by the partners, their attorneys and their financial advisors, to determine whether a need exists for partnership life insurance, and if so, how large a program is necessary.

DENVER WINTER MEETING

By Leonard W. Larson, M.D.
President, American Medical Association

The 15th annual American Medical Association clinical meeting in Denver Nov. 26-30 will offer a combination of fundamental postgraduate knowledge plus the latest findings in a number of areas of medical research that will be of great benefit to all of us in the conduct of our practice.

As a former member for many years of the Council on Scientific Assembly, I have followed the progress and development of the winter clinical meeting from its inception. I can state without qualification that the program organized for this 1961 Denver meeting is the best that has ever been assembled.

At the annual meeting in New York last June, the Board of Trustees and the House of Delegates once again put their stamp of approval on the winter clinical meeting as a vital part of the American Medical Association's service to its membership to provide continuing education and knowledge.

It is my personal hope and appeal that every member of the American Medical Association will take full advantage of the opportunities offered at the Denver meeting by attending all five days.

There are many highlights in the clinical programs that will be of value and interest to the clinician.

All of us in practice are well aware that the personal habits of our patients, plus the habits of the social group of which they are a part, play a major role in health.

This phase of medicine has been studied in detail by a group of Colorado physicians, and they will present their findings in a series of papers at the Denver meeting.

Space medicine is very much in the news these days. Many of us are only vaguely aware that the research specialists in space medicine also are learning much that will be of value to the physician in everyday practice. Several specialists in space medicine will present papers analyzing some of these findings.

Every physician knows that heredity is important in tracing the patient's pattern of disease. The research scientists are now learning much more about this important aspect of medicine, and a section on genes and chromosomes and their implications in disease has been scheduled.

It is now possible to get bids and delivery dates on a full-fledged nuclear power plant for private industry. In fact, at least one of these plants already has been built. In the decade ahead there will be many more nuclear reactors in everyday use in many geographical areas.

Every possible safety precaution is taken in the installation and operation of a reactor, but there always is the human element, and accidents will happen. The physician in practice, sooner or later, likely will be faced with the problem of treating injuries from reactor accidents.

Specialists in this area will present several papers that will give those of us in practice considerable basic knowledge on how to treat patients suffering from reactor accidents.

I have listed only a few of the many highlights of the clinical program for the November meeting. There will be many other equally interesting and informative presentations.

The winter meeting is designed specifically for the clinician in practice. Let me repeat: the program this year is the best in the splendid history of this meeting.

The Annual Meeting of the
IOWA MEDICAL SOCIETY
will be held at
Veterans Memorial Auditorium
Des Moines
May 13-16, 1962
Please mark you calendar

SHORTCOMINGS OF THE BRITISH NATIONAL HEALTH SERVICE

Just available from Great Britain is the first in a series of pamphlets prepared by John and Sylvia Jewkes. This first publication deals with the National Health Service. Others will examine the medical systems of Britain, the U. S., Canada, New Zealand and Switzerland.

Professor Jewkes, who holds the chair of Economic Organization at the University of Oxford and was a member of the Royal Commission on the Remuneration of Doctors and Dentists, and his wife find that many Britons believe that medical services in the U. S. are so expensive that nobody can afford to be ill. And, "conversely, there are many Americans who talk of British 'socialized medicine' as a system in which hordes of patients, broken in spirit, resignedly accept heartless treatment from a medical profession degraded and humiliated by a ruthless government department —a picture which, of course, most English people find wholly ludicrous."

After carefully noting the conditions in England and in the U. S. before and after the beginnings of the National Health Service, the Jewkes couple say, "It is difficult to escape the conclusion that in the U. S. the quantity of medical services available is larger and is tending to increase more rapidly than in Britain." In 1939, they point out, Great Britain was more amply supplied with hospital beds than the U. S. But England has built few hospitals since 1947 while, under the Hill-Burton Act, the U. S. has averaged 150 new hospitals each year. And since 1921, the number of American physicians per thousand population has consistently been higher than in Great Britain.

Perhaps the only place in which Britain excels in personnel is with nurses. In relation to population there have always been more in England.

Professor Jewkes is particularly critical of the administration of the National Health Service which, in seeking to impose a "central pattern and purpose" on the medical services as a whole, has built up a complex hierarchical administrative structure which, in its turn, is now being criticized as productive of delay and confusion. Conversely, many recent reports have stressed the virtues of slow and organic growth, and of smaller and heterogeneous groupings, and warned of the danger that too firm a grip on policy at the center may enfeeble actions at the periphery.

Most important in this paper, however, is the discussion of the economics of medical services. Medical services in Great Britain continue to be purchased privately, and about one half of all the pharmaceutical products consumed are purchased privately. Voluntary health insurance has grown rapidly in Great Britain because people are ready to make sacrifices in other directions in order to enjoy prompt hospital and specialist treatment, free choice of specialists, and private accommodations.

Finally, the Jewkes pamphlet suggests that the National Health Service may have "positively hindered the growth of British medical service."

On this, the editor of the BRITISH MEDICAL JOURNAL comments: "We may or may not agree with this, but at least the remark comes as a challenge to those who boost Britain's National Health Service as if it were one of the most remarkable things that has happened in the twentieth century. The same unctuous self-praise—that the National Health Service is an example to the world —may well be 'the kind of pretentious claim likely to be indulged in by a power which finds its place in the world slipping, does not relish it, and seeks compensation in national day-dreaming.' "

—MORRIS FISHBEIN, M.D., in MEDICAL WORLD NEWS

RECOMMENDATIONS FOR SAFER FOOTBALL HELMETS

The hard plastic football helmet with protruding face guard and "knife-like" rear rim was criticized in the August 12 issue of J.A.M.A. by Richard C. Schneider, M.D., Fritz Crisler and Bennie Oosterbaan, of the University of Michigan. They reported on a study of 14 fatal injuries to the head and spinal cord in 1959 and several others in 1960, saying that they had been prompted by "a slowly progressive upward trend" in football injuries involving the head and spinal cord since 1947.

They urged the use of more resilient materials in the helmet, the removal of, or changes in, the plastic face guard and improvement in the chin strap.

"Some coaches have recognized that the protruding face bar may be responsible for serious injuries," they said. "With its introduction, neck injuries have increased in number." It represents a handy lever, and in professional football it is permissible to grab the face guard in tackling. The face guard also has been shown to hinder the player's vision, whereas his ability to see an opponent who is about to block or tackle him can prevent injury, they declared.

"The face guard, *if it continues to be used* and is made of solid plastic material, should be shortened and placed closer to the face," the authors advised. "This would provide the following advantages: (a) a less accessible bar for the opponent to grab; (b) less leverage available if backward thrust is employed; (c) better vision due to less reduction in visual field; and (d) less overriding surface of the bar, which might injure an opponent's face.

The firmly-tightened chin strap was also criticized. "There may be a place for the development of a chin strap which would release at certain

predetermined pressures, like the safety bindings on skis," they said.

"The remarkable thing about such an investigation," they concluded, "is that considering the vast number of participants in football, there are only an infinitesimal number of fatal injuries."

MENTAL ILLNESS STRIKES ONE IN EIGHT URBAN RESIDENTS

Approximately one in eight urban dwellers (12.5 per cent) is suffering " a more or less serious mental disorder at any given point in time," according to Dr. Benj. Pasamanick, a professor of psychiatry at Ohio State University, in the August issue of ARCHIVES OF GENERAL PSYCHIATRY.* "About one in 40 (2.6 per cent) is severely or totally impaired by mental or emotional difficulties at any one time."

Dr. Pasamanick based his statements on a series of studies, conducted in Baltimore, which he said "constitute a relatively complete assortment of the reported, unreported, and publicly institutionalized cases of mental disorder." The data studied included records of private and public institutions and a survey of a sample of more than 800 non-institutionalized Baltimore residents, he said.

Although one in eight is a lower ratio than has sometimes been reported, it is much higher than the frequently quoted one in ten figure and other rates based solely on reported and recorded cases The one in ten figure is based on a study which excluded institutionalized patients, he said.

The Baltimore studies indicated that 7 to 8 per cent of the mental cases were psychotic and about 15 per cent mentally deficient. More than 75 per cent could be classified as psychoneurotics and persons with character-trait disturbances or psychosomatic types of disorders, Dr. Pasamanick said.

* Pasamanick, B.: Survey of mental disease in urban population: IV. approach to total prevalence rates. ARCH. GEN. PSYCHIAT., 5:151-155, (Aug.) 1961.

AMA CONFERENCE ON TREATMENT OF MENTAL ILLNESS

In preparation for a concerted campaign against mental illness and for better mental health, a planning conference will be held in Chicago September 29-October 1, under the auspices of the AMA. Some 150 key individuals knowledgeable in specific areas of mental health and illness will attend it. There will be no representatives from organizations as such.

The objectives of the conference will be to assess the current activities in mental health, to bring the problem into focus, and to consider procedures for the effective prevention and management of mental disorders.

The conference will seek to identify areas and methods where and by which physicians and the AMA can provide additional support and can catalyze the development of community and hospital services, research, and professional and lay education. The resultant program will be presented afterward to mental health representatives of state medical societies for their consideration and discussion. A group of those state society representatives is tentatively scheduled to meet in January or early February.

One of the first major activities of the new mental health campaign will be an AMA Congress on Mental Health, to be held in cooperation with the American Psychiatric Association. That meeting is tentatively scheduled for June, 1962, in Chicago.

How Fast Did You Say You Could Stop?

It takes the usual good driver three-fourths of a second to achieve actual brake pressure. See the table below for the distance your car travels while you are moving your foot from the accelerator to the brake pedal.

Now, as regards stopping distance—after you have applied your brakes. The figures given here are for DRY pavement and for perfect tires and brakes. For cars with regular tires, multiply by *three* for snow; multiply by *ten* for ice. To get effective braking on ice, reinforced chains, with a welded crossbar on each link, must be used. Even with such chains, the stopping distance is more than three times the distance on dry pavement.

Note that the braking distance increases as the square of the speed increases—i.e., the braking distance at 60 m.p.h. is nine times the braking distance at 20 m.p.h.

Speed		Stopping Distance		
Miles Per Hour	Per Feet Second	Distance Traveled During Reaction Time	Braking Distance	Total Stopping Distance (in Feet)
20	29	22	21	43
30	44	33	47	80
40	59	44	84	128
50	73	55	131	186
60	88	66	188	254
70	103	77	268	345

Iowa Chapter of the American Academy of General Practice

MANAGEMENT OF AAGP AFFAIRS

A frequent criticism of medical organizations is that the individual member has nothing to say about policy and actions of the organization on a national or even on a state level. This cannot be held true of the Iowa Chapter of the American Academy of General Practice, for each member who attends the state business meeting has a vote and the right to challenge the actions of the executive committee on the floor. Delegates to the national convention are chosen from this membership at large, as are the state officers.

Each year these state officers meet with those from other states in Kansas City at the State Officers Conference (The SOC) in order to exchange their knowledge and experience with one another, as well as to receive advice and suggestions from the Board of Directors. Regional problems are brought out, and a general educational process takes place, and each state chapter can benefit from the knowledge gained in other states. This meeting for the state officers is strictly for administrative problems, and the organization of the AAGP is no better illustrated than at this meeting.

The overall policies of the Academy are determined by the meeting of delegates and they are then administered by the Executive Committee and Board of Directors, but each state has its own set of officers to run its local affairs. The local officers' duties include setting up an office for direct administration of problems, recruiting new members and sponsoring postgraduate meetings. These state officers reflect the attitude of the whole state and serve as intermediaries between the individual member and the higher administrative level of the national group.

Various specific aspects of the general problems of medicine are covered, and each year a report is made on the legislation in Washington. In 1960 there were general discussions, and at the meeting in September, 1961, Austin Smith will discuss the pending legislation in Washington. The growing problem of medical pre-payment is also discussed each year, and a symposium will be held with representatives from the AMA, Blue Shield, Blue Cross, and private health insurance institutions. Inter-professional relations are also covered in great detail, especially those between the General Practitioners and Specialists.

Publicity problems are discussed in great detail, and proper relationships with the press are also stressed. Probably most useful of all the sessions are those in which the officer from each state can bring up their specific problems and get answers from those who specialize in those matters. More than anything else, these meetings give the officers from each state a chance to see the highly organized, highly efficient office of the American Academy of General Practice in action.

PSYCHIATRIC CONDITIONS AND TECHNICS

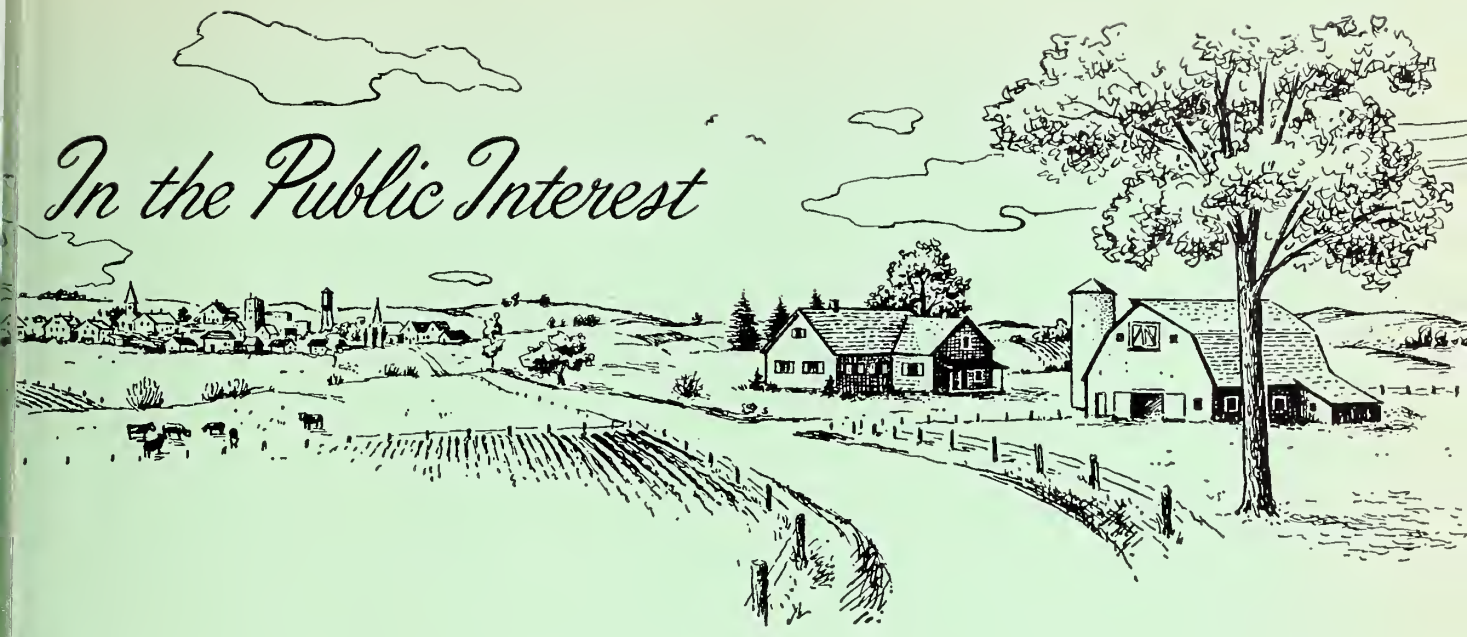
Much effort is being exerted to give non-psychiatrist physicians an awareness and understanding of psychiatric conditions and technics, for the potentialities of full cooperation between psychiatrists and other sorts of doctors are tremendous.

With this in mind, the members of the Webster County Medical Society are inviting all interested physicians to enroll for a series of psychiatric seminars, which will be conducted according to criteria established by the AAGP Committee on Mental Health and enumerated in the report published by that group in May, 1961.

Meetings will be held regularly, in Fort Dodge, at 8:00 p.m. on the first and third Wednesdays of each month from October, 1961, through May, 1962. They will be open-end sessions lasting a minimum of two hours each. Dr. Paul Kersten, a Fort Dodge psychiatrist will conduct the seminars, and enrollment will be limtied. Appropriately organized courses like this have merited AAGP Category I credit, and such accreditation has been applied for.

For further information, write or call Dan S. Egbert, M.D., president of the Webster County Medical Society, 803 Carver Building, Fort Dodge.

Mark these dates on your calendar:
SEPTEMBER 25, 26, 1961
Annual Meeting and Scientific Assembly
Hotel Savery
Des Moines, Iowa



Iowa Should Continue Providing

Liberal Support for the Mental Health Institutes

Rather than impose a ceiling on psychiatrists' salaries or otherwise limit the advances that are being made at the four mental health institutes, Iowa should continue offering attractive salaries to top-notch doctors and should employ additional ones of them just as rapidly as they become available. By making that choice, the state will enable its hospitals to provide increasingly effective treatment for more and more patients, and to return them more promptly to their homes as potentially productive and happy people.

We shan't attempt to prove that employing more and better-trained personnel, during the past few years, has actually saved considerable amounts of money. There is no doubt (1) that since 1950 the average number of patients at Mt. Pleasant, Independence, Cherokee and Clarinda has been reduced from 6,127 to 3,708, or 40 per cent; (2) that overcrowding has thus been completely eliminated at all four institutions; and (3) that the average patient's stay has been reduced from 4.5 to 1.5 years. But it would be ridiculous for us to imply that resultant savings totaling millions of dollars are ready and waiting to be put to other uses.

Overcrowding at the mental health institutes was remedied initially by sending many senile patients to county homes for long-term custodial care, but the bulk of those moves took place in 1949-1950. Now, only about 300 such transfers are being made each year—a number that the county institutions can easily accommodate—but the percentage of chronics at the state hospitals is continuing to fall. Discharge rates are remaining high in consequence of the intensive treatment that is being provided to patients with acute disorders, and today about seven out of 10 individuals leaving the hospitals are returning to their own homes.

The problems that remain are major ones. As discharge rates at the state mental hospitals have risen, readmission rates have increased almost as steeply and consistently, while first-admission rates have at least remained constant. It has been pointed out that by no means all "readmissions" represent relapses or failures of therapy. A peculiarity of hospital bookkeeping requires that entering patients shall be classified as readmissions if they ever have been enrolled at a psychiatric facility of any sort. Thus, for example, all patients who have undergone diagnostic studies at Broadlawns-Polk County Hospital, in Des Moines, and have subsequently been referred to Clarinda are classed as readmissions on their arrival there. But however that may be, it is obvious that the enrollments at the four state hospitals aren't likely to decline much further in the foreseeable future.

THE SITUATION WAS BAD ONLY A DECADE AGO

Satisfaction can be taken from the fact that the four mental health institutes are now caring for precisely the numbers of patients that they were originally intended to serve; from the fact that the patients now are more contented than they used to be, and more readily susceptible to treatment; and from the fact that far more and better therapy is being provided them.

In the CEDAR RAPIDS GAZETTE for March 6, 1949, Mr. Loyal Meek reported upon a visit he had made to the Independence Mental Health Institute together with Dr. Charles C. Graves, then the director of state mental institutions. At that time, he said, the buildings which had been built

to serve 1,200 patients contained 1,703 of them. As many beds as possible had been crowded into each of the wards, and most of the patients literally had nothing to do. Two occupational therapists had just been employed, but in each of the rocking chairs that lined the halls in the women's section, a patient rocked all day long, or just sat. Dr. Max E. Witte, who had become superintendent in January of that year, was the only fully-qualified psychiatrist among the seven physicians on the staff.

If Dr. Witte and the three doctors with European psychiatric training could have spent eight hours per day, five days per week administering psychotherapy, each patient's fair share of treatment time would have been just one hour every two months. And since the doctors had admissions examinations to give and other administrative tasks to perform, a patient who received psychotherapy once every three months was fortunate.

Of the 190 other employees at Independence in 1949, very few had had formal training for their jobs. According to Mr. Meek, there were no registered nurses there at all!

Men who had been adjudged mentally unable to stand trial for crimes of which they were accused received no attention from psychiatrists. Up until 10 years ago, doctors from the mental health institutes spent four hours at the Anamosa reformatory twice each year examining unconvicted prisoners. They treated none of them.

HOSPITAL CARE AND TREATMENT HAVE BEEN IMPROVED

The tabulation that appears below provides some objective data on the improvements that have taken place in staffing during the past few years.

STAFF MEMBERS AT THE FOUR MENTAL HEALTH INSTITUTES

	June 30, 1950		*June 30, 1955*		*June 30, 1961*	
	NUMBER	RATIO	NUMBER	RATIO	NUMBER	RATIO
Psychiatrists-Physicians	29	211	35	149	60	66
Psychologists	3	2,042	13	402	23	164
Social Workers	13	471	13	402	43	89
Other Therapists	58	127	56	93	121	33
Registered Nurses	20	306	25	209	89	42*
Attendants-Aides	592	10	840	6	934	4*

* Since nurses and attendants-aides have begun working a 40-hour week, the ratios for 1961 aren't fully comparable with those for previous years for those types of employees.

Intensive psychotherapy can now be given to patients, and it is increasingly effective because English is the native tongue of a larger proportion of the psychiatrists. It isn't our intention to belittle either the abilities or the educations of the foreign-born physicians, but communication between doctor and patient is more important in psychiatry than in any other branch of medicine, and it seems certain that as the European doctors collaborate with larger numbers of American-born physicians, their familiarity with the English language, with Midwestern idiom and with the connotations of their patients' statements must be growing rapidly.

Perhaps the longest forward step at the mental health institutes has been the establishment of in-service training programs. Through the resident-physician programs at Cherokee and Independence, the state is availing itself of the services of psychiatric residents—all of them fully-qualified doctors of medicine—who are learning their specialty under the supervision of highly skilled teachers. Courses in mental health problems for nurses and other types of personnel have also been started.

EXTENSION SERVICES ARE NOW BEING STAFFED

The staffs at all of the mental health institutes will need some further expansion if maximum therapeutic efficiency is to be attained, but the current director of state mental hospitals, Dr. James O. Cromwell, wishes next to furnish consultative services to communities by locating psychiatrist-psychologist-social worker teams throughout the state. His department is responsible for the provision of requisite therapy to the patients who have been sent from the institutes to county homes, and in addition he wants professional workers from the hospitals to consult with the personnel of mental health clinics and the psychiatric departments of general hospitals about preventive programs and about after-care for mental patients who have been returned to their homes. He will need to add more psychiatrists and psychologists to his staffs for those purposes.

A psychiatric division independent of the state mental health institutes has been established at Anamosa to serve criminally insane patients from the reformatory and from the state penitentiary at Fort Madison. Male mental patients are also transferred there from the state schools for the mentally retarded and other Board of Control institutions. Consultation service only is provided to the Women's Reformatory at Rockwell City and the State Soldiers Home at Marshalltown. A psychiatric team is stationed at Fort Madison as well, and a third team at Eldora renders psychiatric service to the training schools there and in Mitchellville, and to the State Juvenile Home at Toledo.

In formulating his plans and in implementing them, Dr. Cromwell has had the advice and the full support of the IMS Committee on Mental Health, and it is his firm belief that the program will do a great deal to foster the private practice of psychiatry and clinical psychology throughout the state. The efforts of the state hospital people in strengthening both preventive and after-care services will help awaken the public to the fact that mental disturbances, like other illnesses, must be treated promptly and efficiently. When that general change of mind has taken place, Dr. Cromwell is confident that greater numbers of psychiatrists and psychologists can be expected to locate in major towns throughout Iowa.

Iowa Association of Medical Assistants

NATIONAL MEDICAL ASSISTANTS' MEETING

"Reaching For The Heights" is the theme of the Fifth Annual Convention of the American Association of Medical Assistants to be held in Reno, Nevada, October 13-15, 1961. The registration fee of $25.00 includes luncheons and banquets.

The Board of Trustees will meet two days prior to the Convention at Lake Tahoe, and festivities for members and delegates will begin on Thursday evening, October 12, with a reception at the Holiday Hotel from 7:00 to 10:00 p.m.

October is a beautiful month in Nevada, with colorful, warm days and cool evenings. For those who have some vacation time, there will be several tours available: Beautiful Lake Tahoe; scene of the Winter Olympics, Squaw Valley; Virginia City; California; and Hawaii.

Friday, October 13, 1961

8:00 a.m. Registration

9:00 a.m. General Session—Bettye Fisher, Presiding
Greetings and Introductions

10:00 a.m. House of Delegates—Lucille Swearingen, Speaker
Swap Shop, "To Reach The Mountains"—Mary Kinn, Moderator
1. "Not only to have, but to hold"—Edith Jones
2. "From Studying the Hills, I learn of the Mountains"—Lois Pluckham
3. "Conservation of the Natural Resources"—Kathy Rand
4. "Sunrise and Fresh Ideas"—Marian Little

12:30 p.m. State Luncheon—Margo Fry, Nevada, presiding
Speaker: James R. Greear, M.D., president, Nevada State Medical Association

2:00 p.m. House of Delegates
"Define Guilt"

3:15 p.m. Break for Exhibits

3:30 p.m. The Narcotics Problem
Speaker: George H. White, F.B.I.

5:00 p.m. Break

6:30 p.m. Cocktail Party—Courtesy of Harolds Club

7:30 p.m. Dinner
Subject: "Quacks"
Speaker: Oliver Fields, Director, Bureau of Investigation

Saturday, October 14, 1961

8:30 a.m. House of Delegates

9:00 a.m. General Session
Topic: "Who Will Train Tomorrow's Medical Assistant"
Panel: Carol Towner Brierly, AMA
Donna Bills, Utah
Edward Viette, Ph.D., University of Nevada

10:30 a.m. Break for Exhibits

10:45 a.m. Horace Cotton, Medical Economics

11:30 a.m. Break for Luncheon

12:00 Noon Luncheon
Speaker: W. W. Hall, M.D.
Subject: "How Is Your Risorius Working?"

2:00 p.m. Third Annual Leadership Symposium
Established through grant from Lakeside Laboratories
"FOR I DIPT INTO THE FUTURE"
a. New Devices
Speaker to be announced later
b. New Drugs
Speaker: Austin Smith, M.D., President, American Pharmaceutical Association, formerly editor of the JOURNAL OF THE AMA
c. "Oxygen: Our Life"
Speaker: Stanley E. Fey, K-G Equipment Company
d. Space Medicine:
Speaker: U. S. Air Force Officer
e. Future of Medicine:
Speaker: Leonard Larson, M.D., President of AMA

5:00 p.m. Break

8:00 p.m. Banquet—Bettye Fisher, presiding
Speaker: Leonard Larson, M.D., President, AMA

Sunday, October 15, 1961

9:00 a.m. Brunch: Lillie Woods, presiding
All newly elected officers and directors
All State Presidents and Chapter Presidents

10:00 a.m. Orientation of New Officers

11:00 a.m. Panel Discussion
"Will You Put Your Doctor In Jail?"
Moderator: Nelson Neff, Executive Secretary, Nevada State Medical Association
Panel: Howard Hassard, Executive Director, California State Medical Association
William A. O'Brien, III, M.D., Reno, Nevada
Robert L. McDonald, Attorney at Law, Reno, Nevada

12:30 p.m. Adjournment

STATE DEPARTMENT OF HEALTH

Edward G. Zimmer
COMMISSIONER

HUMAN BRUCELLOSIS IN IOWA, 1960

During 1960, Iowa physicians reported 379 human cases of brucellosis. According to preliminary data from the U. S. Public Health Service, these cases comprise about 40 per cent of the cases reported in the United States, For several years, Iowa has had one-fourth to one-third of all the reported brucellosis. Although the numbers of cases in Iowa have declined in the last 10 years, the decline has been greater in other states. Therefore, Iowa's percentage of total cases in the country has increased.

The cases reported for Iowa and for the United States in each of the last 10 years are as follows:

Year	Iowa	United States	Year	Iowa	United States
1951	767	3,139	1956	360	1,300
1952	724	2,537	1957	214	983
1953	556	2,032	1958	283	924
1954	351	1,823	1959	361	892
1955	405	1,444	1960	379	742*

* Tentative—will be increased by delayed reports.

In Iowa last year, 78 per cent of the cases were either farmers or packing house workers. The remaining 22 per cent of the cases occurred among veterinarians, livestock truckers, rendering plant employees, students, housewives and persons in other occupations. This distribution emphasizes the increasing occupational nature of the disease. Cases develop among persons with direct or indirect contact with infected animals or with their fresh tissues. The percentage of cases among packing plant employees was higher than in previous years. Although cases occur among persons classified as housewives and students, most of those people live on farms and have contact with animals. With the increased use of pasteurized milk by farm families as well as by urban residents, human cases due to contaminated milk are few in number.

The following table showing the age distribution of cases likewise emphasizes the occupational nature of the disease. No patients were under 13 years of age.

Age	Cases	Age	Cases
0- 9 years	0	20-29 years	102
10-19 years	14	30-39 years	105
40-49 years	57	60+ years	8
50-59 years	15	Not stated	78

MORBIDITY REPORT FOR MONTH OF JULY 1961

Disease	1961 July	1961 June	1960 July	Most Cases Reported From These Counties
Diphtheria	0	0	0	
Scarlet fever	104	94	99	Jefferson, Johnson, Polk
Typhoid fever	0	0	3	
Smallpox	0	0	0	
Measles	236	892	84	Buena Vista, Des Moines, Scott
Whooping cough	7	9	3	Polk
Brucellosis	29	17	13	Dubuque, Scott
Chickenpox	47	269	131	Clay, Scott, Story
Meningococcic meningitis	1	0	0	Polk
Mumps	127	287	288	Des Moines, Dubuque, Scott, Story
Poliomyelitis	1	0	2	Des Moines
Infectious hepatitis	159	123	20	Boone, Polk, Scott, Warren
Rabies in animals	53	36	17	Dallas, Hardin, Marshall, Muscatine, Story
Malaria	0	0	0	
Psittacosis	0	0	0	
Q fever	0	0	0	
Tuberculosis	35	31	40	For the state
Syphilis	74	77	120	For the state
Gonorrhea	111	132	150	For the state
Histoplasmosis	7	2	0	Black Hawk, Chickasaw, Polk
Food intoxication	0	0	0	
Meningitis (type unspecified)	1	2	0	Cerro Gordo
Diphtheria carrier	0	0	2	
Aseptic meningitis	0	0	2	
Salmonellosis	2	4	7	Jasper, Wapello
Tetanus	1	0	0	Polk
Chancroid	0	0	2	
Encephalitis (type unspecified)	1	0	0	Scott
H. influenzal meningitis	0	1	0	
Amebiasis	2	3	0	Audubon, Johnson
Shigellosis	3	4	2	Black Hawk
Influenza	0	0	0	

DISCOVERIES IN LEUKEMIC-CELL METABOLISM

Joint research by U. S. and Russian scientists at the U. S. Public Health Service's National Cancer Institute has revealed a difference in the way two types of leukemic cells conduct their metabolic, or energy-producing, activity. According to the Cancer Institute, knowledge of this kind is helpful in designing more effective drugs against leukemia.

Dr. Dean Burk of the Institute's Laboratory of Biochemistry reported this finding at a meeting of the Fifth International Congress of Biochemistry held August 10-16 in Moscow. The work was carried out by a group that included Dr. John Laszlo, formerly of the Institute's General Medicine Branch; Dr. Berigoj Stambuk, formerly of the Laboratory of Biochemistry; Dr. Mark Woods, of the Laboratory of Biochemistry; and a Russian investigator, Dr. Joseph F. Seitz, who spent six months at the Institute under a World Health Organization fellowship.

Drs. Burk and Woods have long studied cell metabolism along guidelines proposed some 30 years ago by the German biochemist, Otto Warburg, who held that cancer-causing agents reduce a cell's ability to use oxygen to convert sugar into energy. While normal cells rely almost exclusively on a process requiring oxygen (respiration), malignant cells, according to Warburg, also employ a process needing no oxygen (fermentation).

A few years ago, a group headed by Drs. Burk and Woods was able to attribute the high level of fermentation in malignant cells to a breakdown in the hormonal mechanism that normally controls one step in metabolism. Suspecting that this defect could provide a target for cancer chemotherapy, the group then tested and confirmed the ability of several known anticancer agents to restore the missing control.

The results also furnished evidence of a difference in the rates of fermentation by both normal and leukemic cells of the lymph system and those of the bone marrow in the presence of oxygen. Supporting data were obtained by measuring the fermentation of cells maintained under conditions closely simulating those prevailing within the body.

Though these findings indicate a difference between lymph and bone marrow cells, they reveal no distinction, with respect to fermentation, between normal and leukemic cells. Further studies are under way to determine whether normal and leukemic cells differ with respect to respiration. A finding of such a difference might make it possible to design drugs that would attack leukemic cells only. The tendency of known anticancer drugs to damage normal as well as malignant cells is one of the most difficult problems in cancer chemotherapy.

Drs. Burk and Woods are continuing this work at the Cancer Institute. Dr. Seitz is now at the Institute of Hematology and Blood Transfusion, Leningrad. Dr. Laszlo is now at the Duke University Medical School, Durham, N. C., and Dr. Stambuk at the Veterans Administration Hospital, Chicago.

PROCEDURES FOR OBTAINING LICENSE TO MARRY

FOR PERSONS IN IOWA WHO PLAN TO BE MARRIED IN IOWA

1. An examination, which shall include a blood test, must be obtained by both parties to be married, from a physician licensed to practice in Iowa. (Chapter 596, Code of Iowa, 1958.)

2. The blood test must be performed by the State Hygienic Laboratory at Iowa City or by such other laboratories in the state as are approved by the Iowa State Department of Health. Listings of such approved laboratories are available at each of the offices of the county clerks of court.

3. If the blood specimen is to be sent to the State Hygienic Laboratory at Iowa City, the applicant should allow sufficient time in his marriage plans for mailing of the specimen and return of the results by mail, which is usually three (3) to five (5) days.

4. The examination and blood test must be made within twenty (20) days prior to making application for license to marry.

5. The completed certificate of health examination is to be presented to the clerk of the district court of the county in which the marriage is to be solemnized at the time application is made for license to marry.

6. "Previous to the issuance of any license to marry, the parties desiring such license shall sign and file a verified application with the clerk of court which application either may be mailed to the parties at their request or may be signed by them at the office of the clerk of district court in the county in which the license is to be issued." (House File 223, Act of the 59th General Assembly.)

7. "Such application shall set forth at least one affidavit of some competent and disinterested person stating such facts as to age and qualification of the parties as the clerk may deem necessary to determine the competency of the parties to contract a marriage." (The clerk of court cannot act as the required witness.) (House File 223, Act of the 59th General Assembly.)

8. "Upon the filing of the application for a license to marry, the clerk of the district court shall file the application in a record kept for that purpose, and no license shall be issued until the expiration of three days from the date of filing the application." (House File 223, Act of the 59th General Assembly.)

9. "After the expiration of three days from the date of filing, the clerk shall issue the license to the parties if he is satisfied as to the competency

of the parties to contract a marriage." (House File 223, Act of the 59th General Assembly.)

10. The date of filing is excluded and the last day is included. Thus, if a couple applies for a license on Monday, the clerk of the district court may issue the license at any time Thursday. (See O.A.G. June 27, 1961, Straus to Skiver, County Attorney.)

11. The license to marry may be obtained by either of the parties to a marriage. Both applicants need not be present. The marriage license cannot be issued by mail. (See O.A.G. June 27, 1961, Straus to Skiver, County Attorney.) The license fee is three dollars ($3.00).

12. Marriages must be solemnized within twenty (20) days following the issuance of license to marry in the county of issuance by an authorized person such as a justice of the peace, judge or ordained or licensed minister of the gospel.

13. At least two witnesses must be present at the time and place of the marriage.

14. Minimum age requirements for marriage in Iowa are twenty-one (21) for the male and eighteen (18) for the female. Males between eighteen (18) and twenty-one (21) and females between sixteen (16) and eighteen (18) may marry with written consent of parents or guardian.

15. "The District Court may, when application is made by parties one or both of whom are under the age thus fixed and the female is pregnant, grant an order authorizing issuance of a marriage license by the clerk of the district court to said applicants, and the marriage under such license shall be valid. The records of the court which pertain to such conditions of pregnancy shall be sealed and available only to the contracting parties or to any interested party securing an order of court." (House File 269, Act of the 59th General Assembly.)

FOR PERSONS IN IOWA WHO PLAN MARRIAGE IN ANOTHER STATE

1. Premarital examination requirements of other states may be satisfied by having the physician send a blood specimen to the State Hygienic Laboratory at Iowa City. The physician should write on the face of the laboratory slip which accompanies the specimen or attach a note to the effect that the applicant plans to marry in another state, and should give the name of that state. The State Hygienic Laboratory will send an examination certificate on the proper state's form for the physician to complete if the Iowa forms are not acceptable. The full name of the patient, street, street number and city must be given.

2. Other requirements such as waiting period, minimum age requirements, who may make application and period of time blood test is honored, vary from state to state. Therefore, it is suggested that the applicant write for this information to the state department of health of the state in which the marriage is to take place, or to the State Hygienic Laboratory at Iowa City.

FOR PERSONS NOT IN IOWA WHO PLAN TO MARRY IN IOWA

1. An examination, which shall include a blood test, must be obtained by both parties to the marriage from a physician licensed to practice in the state where the applicant lives. (Sec. 596.8, Code of Iowa.)

2. The blood test must be performed by the state health department's laboratory of the state where the applicant lives, or by a laboratory of the Public Health Service or Armed Forces or laboratories of the health departments of New York City, the District of Columbia, or the official provincial health departments of Canada.

3. The official form of the state, district or province is acceptable in Iowa, provided that the state has a blood test requirement comparable to that of Iowa. Residents of states not having comparable blood test requirements must use the Iowa Non-Resident form.

4. The new Iowa law permits application for marriage license by mail. Application should be made directly to the clerk of court in the county in which the mariage is to be solemnized. When the application form has been completed, it is to be returned to that clerk of court.

5. When the clerk of court has received an application by mail and the affidavit is properly executed and acceptable to the clerk of court, the applying parties do not have to have another witness present when they go in to pick up their marriage license. (The marriage license cannot be issued by mail.)

6. Minimum age, time requirements, etc. are the same as for persons living in Iowa, as listed in paragraphs 4 through 15 of the first section of this outline.

MARRIAGES PROHIBITED

1. Marriages contracted within less than one year after either party has been divorced from a former spouse are illegal unless permission has been granted by the court in such decree, when divorce has been granted in the State of Iowa.

2. Marriage is prohibited if either party is disqualified from making any civil contract. (Sec. 595.3, Code of Iowa.)

3. Marriage is prohibited if the parties are within the degrees of consanguinity or affinity enumerated in Sections 595.3 and 595.19, Code of Iowa.

4. Marriage is prohibited if either party is mentally ill or retarded, a mental retardate. (Section 595.3 as amended by Section 184, Chapter 152, Acts 58th General Assembly.)

The above information has been prepared by the Iowa State Department of Health and approved by the office of the Attorney General of the State of Iowa as of July 10, 1961.

Woman's Auxiliary News

OUR PRESIDENT SAYS—

Working without a goal is drudgery. A goal without work is a dream. Working toward a goal is progress.—Martin Vanbee

September finds us back from vacation, our children anxious to return to school and college, and our organizations and clubs resuming their activities. Our county Auxiliaries have an important role to perform each year, and it is no different in 1961-62. You have received your Auxiliary Yearbook with program suggestions, the important Auxiliary dates, and the listing of our county, state, and national officers.

The district meetings are scheduled for September and October. Each physician's wife in the Central Area received a notice and invitation to attend the meeting scheduled in her district. The district meeting has a three-fold purpose. It serves as a social occasion for doctors' wives within the district; it serves as a day of instruction concerning Auxiliary aims and projects rendered by state officers and chairmen of committees; and it provides an opportunity for members to learn of the many county Auxiliary accomplishments.

The meetings are as follows: District V, at Hotel Holst, Boone, September 18, with Boone County Auxiliary as hostess, and Mrs. H. W. Smith, councilor; District X, at the Osceola Country Club, September 21, with Mrs. E. E. Lauvstad as hostess, and Mrs. C. A. Trueblood, councilor; District II in Mason City, September 26, with the members of the Cerro Gordo County Auxiliary as hostesses and Mrs. G. I. Tice, councilor; and District IX, at Knoxville, October 18, with the members of the Marion County Auxiliary as hostesses, and Mrs. L. F. Catterson, councilor.

May the District Meetings this fall prove to have been crowning successes. You who are Auxiliary members, and those of you who are eligible, will have been present and can determine their value. Our success and progress depend upon our attendance, our unity and understanding of purpose, and our individual acceptance of the opportunities and responsibilities afforded.

—Mrs. B. F. Kilgore
President

Many of you will remember Mrs. Keith Chapler, who for several years so efficiently edited the Auxiliary News. You will be interested to know that her son, Christopher K. Chapler, was married to Mary Ann McKowen of Des Moines in Dexter on August 6, 1961.

AS I SEE IT

Organized medicine's stand against socialized medicine is but a part of the whole national conflict concerning our country's "road ahead." Every citizen must help make the choice in plotting the course of this great nation which was built on individual freedoms. We must choose either the welfare state, with questionable security in bondage and no responsibilities, or freedom of enterprise and freedom of the individual, with the resultant responsibilities.

The majority of the medical profession has endorsed a democratic government and the free enterprise system which has allowed our country to grow into the great nation it is today.

Individuals and organized groups who favor a welfare state continually contrive to build our national government from a servant with sharply limited powers into a master with virtually unlimited powers with a multitude of programs touching every phase of our economy and offering us increased "security." Their aim is for all citizens to become wards and dependents of the state, giving government the power to grant or withhold from us the very necessities of life because we are taxed too heavily to pay for them ourselves. To do this they will unceasingly strive to point out to the public every weakness, abuse or so-called inadequate service of the individuals or groups believing in free enterprise.

Such activity is well demonstrated in the drive to change the public image of the doctor. The socialistic attacks on the medical profession have put our husbands on the defensive for too long, and we now must change the content of our programs to a positive offensive. To do this we must first have self-education. Then we must pass accurate knowledge along to all segments of the public. The public accepts the image created for them by publicity and deeds. I refer you to the July 1961 COSMOPOLITAN article "What Is Your Doctor *Really* Worth?" and to the one in the August 1961 GOOD HOUSEKEEPING entitled "What Women Really Think About Their Doctors."

Much of our foreign problem resolves itself into the job of "selling America." To sell America

we must first sell ourselves. Everyone has something to sell, either services or commodities. We need to put a lot more courtesy and enterprise into our selling. These attributes would be wonderful qualities in all our daily lives.

Applied to our husbands' profession, salesmanship is needed to rebuild the public image of the doctor to its former status, selling the public on his professional integrity and ability. We must sell ideas as well as promote activities. Both the patient and the doctor are entitled to respect and courtesy, and as individuals they have special needs and feelings which both should seek to understand. If this objective is to be realized, medical societies and Auxiliaries need 100 per cent participation from all members, and the professional skirts must be kept clean. No persons are better fitted to aids in these activities than the doctors' wives.

As good citizens we must accept and believe that:

Government is everybody's business
Prosperity is everybody's business
Service is everybody's business

We must include this thinking in all our activities.

In our National Program you will find seven activities listed under Legislation. I think it well to include some of the above philosophies as we . . .

Increase and improve group contacts
Distribute pamphlets
Urge other groups to write legislators
Help keep the doctor informed.

One final important point. . . . We are not in this alone. If we expect other groups to work for and with us we must help them attain *their* goals and we must develop an understanding of their problems. . . . THIS IS NOT A ONE WAY STREET.

—MRS. DEAN H. KING, Regional Chairman
Third District Legislative Committee
Woman's Auxiliary to the AMA
307 East Fourth Street, Spencer, Iowa

NATIONAL PARTICIPATION

Mrs. D. H. King, Spencer, has accepted the appointment as Regional Chairman, Third District Legislative Committee, Woman's Auxiliary to the American Medical Association.

Mrs. R. F. Nielsen, Cedar Falls, has been appointed to a Special Committee for Study of International Philanthropic Interests.

Both of the above appointments were made by Mrs. Harlan English, National Auxiliary President, following 1961 meeting in July and in recognition of Iowa's outstanding legislative program with Mrs. King as chairman and co-chairman of that committee at the state level and Mrs. Nielsen's great interest in the leprosy relief project in which Iowa out-performed all other states the past year.

SPEAKING UP FOR MEDICINE

A highlight of the Wednesday morning session at the New York City convention of the AMA Auxiliary, last June, was a presentation by Mr. Dick Reinauer, director of the Radio and Television Motion Picture Department of the AMA's Communications Division. He gave a number of suggestions for improving the Auxiliary's effectiveness in utilizing the various communications media.

He urged his listeners to stand up and speak up for medicine whenever an opportunity offers in women's organizations other than the Auxiliary, and in church groups. The great majority of people, he pointed out, either are uninformed or don't care. It is up to us to inform them of the dangers that socialized medicine poses for all of the American people. The AMA has supported the Kerr-Mills plan of medical assistance for the aged, since it will help only those who really need help. Tax money should not be used to help those who are able to take care of themselves. The basic objectives of the AMA—the promotion of the science and art of medicine, and the betterment of public health—should be impressed again and again upon all of our fellow citizens.

Mr. Reinauer presented several skits designed to show the wrong and the right ways of putting across the Auxiliary's point of view in radio and television appearances. His horrible examples included:

Nervous Nellie, who played with her jewelry, didn't know how to handle the situation, hadn't organized her facts, ignored the interviewer's questions, and stuttered and stammered.

Galloping Gert, who strayed to subjects not pertinent to the interview and let the interviewer take over.

Clothes Horse Hanna, who wore furs and too much jewelry, had a large hat, didn't know the facts and misstated some of them, and had to be corrected by the interviewer.

Forgetful Flossie, who succumbed to nervousness and forgot what she had intended to say.

Bossy Bessie, who was too talkative and tried to monopolize the show.

Mr. Reinauer warned each Auxiliary to be careful, in selecting a representative to appear on TV or radio, to choose a woman who will do justice to the organization—not one of the characters sketched above, but someone who has a good speaking voice, who is well poised and who makes a good appearance. The impression she makes as an individual will be important to the success of the presentation.

Publicity seeks to inform; promotion also informs, but is used to encourage action on behalf of a specific project. Public relations is a combination of the two, plus day-to-day activities designed to build sound relations in the community

—ones that will enhance the group's reputation and its ability to serve.

An Auxiliary that is planning a radio or TV program should have its publicity chairman call the program director of the local station and make an appointment to see him. She should find out whether public service time is available, and what type of programming the station offers in the community.

If the Auxiliary's appeal is to be effective, the publicity chairman should have answers to some key question even before she contacts her local stations. The important questions begin with the words WHAT? WHO? and HOW? WHAT is the Auxiliary's message? WHO should receive that message? Can it be tailored to those whom the Auxiliary particularly wants to reach? HOW can the message best be put across?

Submit all program copy to the program director as far in advance as possible, in triple-spaced typing on only one side of each sheet of paper. For TV appearances, plan to prepare visual aids such as slides or posters, and when you make them, be sure that they are large enough.

When your presentation has been made, send a letter of thanks to the station personnel in appreciation of the help they have given you.

In order to be sure of getting adequate newspaper publicity for her Auxiliary, the publicity chairman should make friends with a reporter or with the publisher. Materials for publication should be channeled within the Auxiliary so that only the publicity chairman contacts the newspaper. Copy must be submitted early enough to make sure that it will be available for publication at the proper time. Remember that news is a perishable commodity and that the newspaper won't want it very much if it is no longer fresh—i.e., if an issue of the paper has appeared since the event took place. If the Auxiliary is planning a banquet, a notice of the event should be in the hands of the editors at least 48 hours in advance. Then, immediately afterward, the paper should be provided a summary of the events that took place there. Publicity chairman should remember that the ABC's of news-writing are to tell WHO, WHY, WHAT, WHEN, WHERE and HOW. News stories should be typed, double-spaced, on one side of 8½ x 11 paper. The name of the organization should appear in the upper corner of the copy, together with the name and phone number of the individual whom the editors should call if they have any questions to ask.

Every Auxiliary member should work within the other local civic, educational, service and church groups to which she belongs, and should be the town crier for organized medicine. She should help her doctor-husband become an active participant in the fight to preserve the free practice of medicine.

—MRS. G. J. MCMILLAN, Councilor, District VIII

MEMORIAL

As we pause in this hour to pay tribute to these doctors' wives and members of the Auxiliary who are no longer with us, our hearts are saddened. There are other thoughts which come to our minds. We are grateful for their lives, for what they have meant to their families, their friends, their communities and our Medical Auxiliary. We are moved by the solemn realization of man's divine heritage, and the high destiny to which he may attain.

"Our birth is but a sleep and a forgetting;
The soul that riseth with us, our life's star,
Hath elsewhere had its setting and cometh from afar;
Not in entire forgetfulness, and not in utter nakedness
But trailing clouds of glory do we come from God who is our home."

Mrs. F. W. Mulsow, Cedar Rapids (past president)
Mrs. E. T. Warren, Stuart (past president)
Mrs. F. X. Cretzmeyer, Emmetsburg
Mrs. O. A. Elliott, Des Moines
Mrs. L. O. Ely, Des Moines
Mrs. G. A. Field, Des Moines
Mrs. H. J. Heusinkveld, Clinton
Mrs. G. A. Huntoon, Des Moines
Mrs. Josiah Johnston, Des Moines
Mrs. R. H. Meng, Clarinda
Mrs. L. A. Taylor, Ottumwa
Mrs. W. M. Walliker, Clinton
Mrs. E. B. Winnett, Des Moines
Mrs. L. F. Steffens, Dubuque
Mrs. E. C. Petersen, Atlantic

The above memorial was conducted by Mrs. J. L. Kestel, Waterloo, at the Auxiliary annual meeting, April 1961, honoring those members deceased in the year 1960-1961.

SCHOOL PUBLIC RELATIONS WORKSHOP REPORT

Minister (to S. S. Class): How many of you want to go to Heaven?

(Everyone raised his hand except one small boy.)

Minister: Don't you want to go to Heaven?

Boy: No.

Minister: You mean when you get old, you don't want to go to Heaven?

Boy: Oh sure, I want to go then! I thought you were drumming up a load to go today!

This story describes the attitude of those of us who attended the workshop at State College of Iowa (formerly Iowa State Teachers College), July 12-15. We were most indifferent to the "Trump Plan" when we were in the infancy stage of our knowledge of it. As our information and understanding grew, so did our enthusiasm.

No brief summary could possibly do justice to what transpired at workshop sessions, lectures, programs, displays and social gatherings. Of necessity this article can only skim the surface and leave you stimulated to find out what it's all about.

The Discussion: Trump or No-Trump.

Basis for Discussion: "Images of the Future" by J. Lloyd Trump.

Essentially apparent is the inevitability of changes in our secondary schools. The Trump Plan provides for an orderly but drastic change in our schools as we know them today. The Trump school will teach children to be self-sufficient rather than dependent on others for direction. The method is three pronged:

1. Instruction of large groups.
2. Discussion in small groups.
3. Individual study.

The student moves at his own pace. He is given as much supervision as he requires, while being taught to need little supervision.

The Trump Plan will affect the architecture of our school buildings, the use of teachers' and students' time, and technics, methods and devices used in teaching. Some casualties in this new school will be lockers, bells signaling fixed time periods, and large "study halls."

If your community is contemplating new construction of schools, be sure your superintendent of schools is well versed in the Trump Plan. Visit with the school board members in your community to be sure they are aware of these changes in teaching mechanics. Dr. Brimm made the classic statement: "What is educationally sound must be administratively possible." Exciting and wonderful changes are imminent. As citizens, as doctors' wives, as parents, we should be informed of these trends.

The workshop was under the able direction of Mr. George Holmes of the public relations department of S.C.I. We received a wealth of material which the Auxiliary members may borrow. It would be splendid if every Auxiliary could devote a meeting to a description and discussion of the Trump Plan. (Did you guess? No bridge is involved.)

Cedar Falls will have her first "Trump School" as an experiment this fall.

—LILLIAN NIELSEN
(Mrs. R. F. Nielsen)

DO YOU JUST BELONG?

Are you an active member
The kind that would be missed?
Or, are you just contented
That your name is on the list?

Do you attend the meetings
And mingle with the flock?
Or, do you stay at home
To criticize and knock?

Do you take an active part
To help the work along
Or, are you satisfied
To only just belong?

Do you ever go to visit
A member who is sick?
Or leave the work to just a few
Then talk about the "Clique"?

Think it over, member,
For you DO know right from wrong!
Are you an ACTIVE member?
Or, do you just belong?

JOURNAL of The IOWA MEDICAL SOCIETY

IN THIS ISSUE:

- A Symposium on Low Back Pain

OCTOBER, 1961

IN CERTAIN MENINGEAL INFECTIONS effective cerebrospinal fluid levels— effective antibacterial action

CHLOROMYCETIN

(chloramphenicol, Parke-Dav

In the management of certain meningeal infections, CHLOROMYCETIN offers uniqu advantages. It has been described by one investigator as "...the best chemothe apeutic agent for patients with *H. influenzae* meningitis...."[1] In comparative *in vit* studies,[2] CHLOROMYCETIN showed the "highest effectiveness" against *Hemophil influenzae, Diplococcus pneumoniae,* streptococcus, and numerous other pathogen Another report states: "Chloramphenicol is regularly detected in the cerebrospin fluid when blood levels greater than 10 micrograms per ml. are reached."[3] Blood leve of this magnitude are easily attainable with the administration of CHLOROMYCETIN b either the oral or parenteral routes.

CHLOROMYCETIN effectively penetrates the blood-brain barrier;[3-6] provides effecti action against *H. influenzae*[1-4,7-9] and other invaders of the meninges.[5,7,10,11] Produ forms are available for administration by the intravenous, intramuscular, and or routes. For these reasons, CHLOROMYCETIN has contributed conspicuously to th dramatic drop in mortality rates in meningeal infections caused by *H. influenzc* and other susceptible microorganisms.

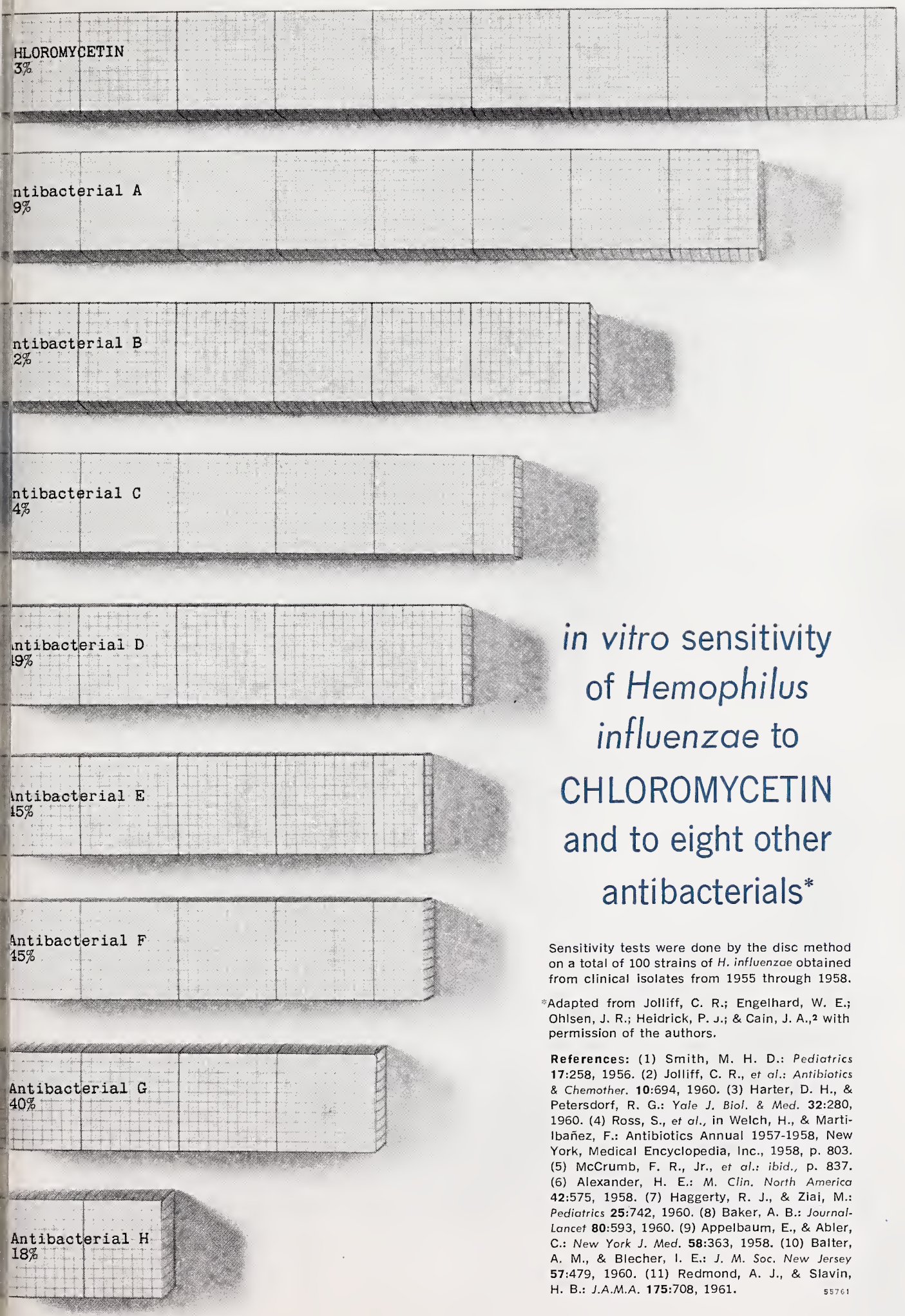
CHLOROMYCETIN (chloramphenicol, Parke-Davis) is available in various forms, including Kapseals® 250 mg., in bottles of 16 and 100. See package insert for details of administration and dosage.

Warning: Serious and even fatal blood dyscrasias (aplastic anemia, hypoplastic anemia, thromboc topenia, granulocytopenia) are known to occur after the administration of chloramphenicol. Bloo dyscrasias have occurred after both short-term and prolonged therapy with this drug. Bearing in mind th possibility that such reactions may occur, chloramphenicol should be used only for serious infectio caused by organisms which are susceptible to its antibacterial effects. Chloramphenicol should not b used when other less potentially dangerous agents will be effective, or in the treatment of trivial infe tions such as colds, influenza, or viral infections of the throat, or as a prophylactic agent.

Precautions: It is essential that adequate blood studies be made during treatment with the drug. Whi blood studies may detect early peripheral blood changes, such as leukopenia or granulocytopenia, before they become irreversible, such studies cannot be relied upon to detect bone marrow depression prior to development of aplastic anemia.

PARKE-DAVIS

PARKE, DAVIS & COMPANY, Detroit 32, Michi



in vitro sensitivity of *Hemophilus influenzae* to CHLOROMYCETIN and to eight other antibacterials*

Sensitivity tests were done by the disc method on a total of 100 strains of *H. influenzae* obtained from clinical isolates from 1955 through 1958.

*Adapted from Jolliff, C. R.; Engelhard, W. E.; Ohlsen, J. R.; Heidrick, P. J.; & Cain, J. A.,[2] with permission of the authors.

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Scientific Articles

Examination of the Back for

Evaluation of Low Back Pain

MICHAEL BONFIGLIO, M.D.
IOWA CITY

ONE OF THE SYMPTOMS encountered most frequently in orthopedics, and in medicine generally, is low back pain. Most physicians have had to deal with the symptom in a patient, or at some time or other have experienced it in themselves. An attempt will be made to demonstrate an approach to the evaluation of a patient with low back pain. The aim of this approach is to permit as thorough an examination as possible, as well as the most efficient use of one's time.

HISTORY

It is axiomatic that an adequate history should be taken before the examination is performed. It is important that information be gathered about past occurrences so that the examiner's attention may be directed to the areas requiring special attention, and so that previous events can be correlated with the physical findings. For example, a patient who has been troubled for many years by low back catches of an acute and sudden type in a slightly flexed position may have a facet syndrome.

The history should do four things: (1) It should evaluate the pain as to location, mode of onset, character and severity, and as to the circumstances in which it is either relieved or aggravated. Frequently, the patient will fail to mention the effect produced upon the symptom, in most instances, by coughing, sneezing or straining during a bowel movement. The physician should then ask those questions. The location of deep pain in the midline low in the back will help the doctor distinguish disc and facet joint disturbances from leg and muscular-strain pain, which may be diffuse in the lumbar region or along the iliac crests where the lumbar fascia attaches. Pain radiating into the buttock posteriorly ("into the hip," as the patient is likely to say), or down the posterior thigh, calf, leg, and foot and toes may be of the dull, aching type. If so, the pain may be either reflex in origin, resulting from the irritation of a posterior spinal nerve ending, or a direct anterior root irritation or compression. In the reflex type, no neurologic deficit is found, but in the direct or true sciatica, there frequently is one. Backache with reflex sciatica is due to mechanical causes or strains, whereas with true sciatica one thinks of disc rupture, disease processes or tumors. A sudden onset of pain secondary to a lifting strain may be the result of disc herniation, a compression fracture of a vertebral body, or a facet asymmetry. Pain that is aggravated by activity and relieved by rest makes one think of mechanical causes, and pain that is more or less constant, and present at night and at rest, leads one to think of a disease process such as metastatic tumor, infection or osteoporosis, although a free-fragment disc rupture may also give pain when the patient is at rest.

(2) The second thing the history should do is to determine the presence of a deformity, limp, list or scoliosis.

(3) The history should provide data regarding neurological symptoms such as sciatic radiation, numbness, tingling and weakness.

(4) It should ascertain the course of an injury or an illness—its mechanism of occurrence, and

Dr. Bonfiglio is a professor of orthopedic surgery at the S.U.I. College of Medicine. He made this presentation on April 25, 1961, at the IMS annual meeting.

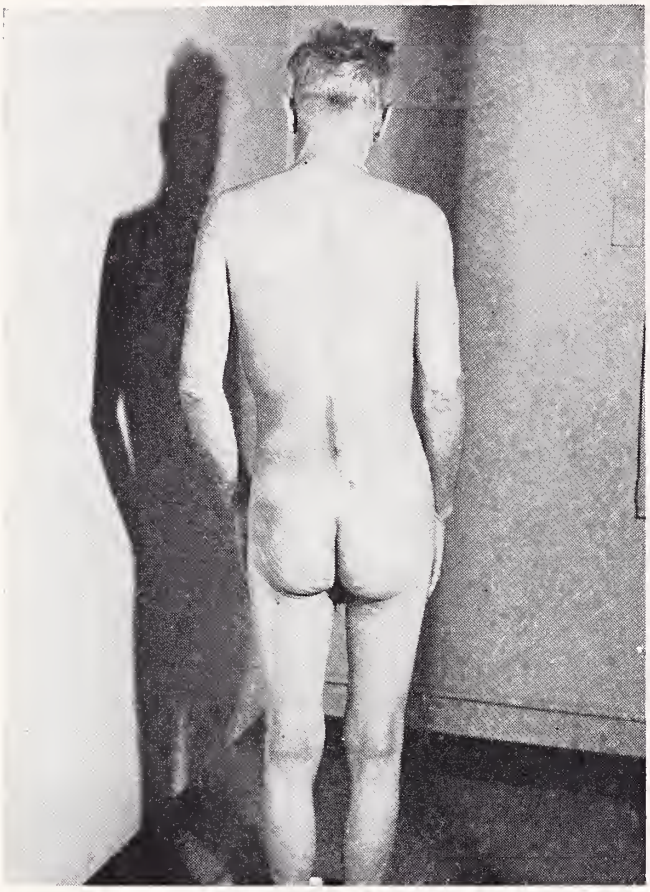

Figure 1. Lumbar list or scoliosis to the left.

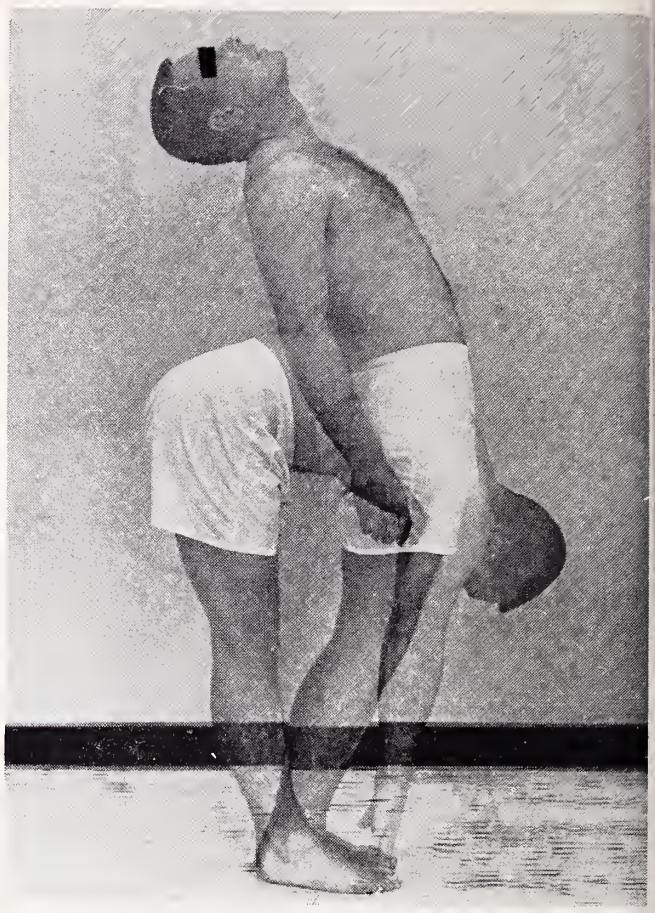

Figure 2. Flexion and extension of the spine.

its chronological relationship to the onset of symptoms. When a patient has related his back pain to an injury, it isn't uncommon to find that the injury actually occurred many years before the symptom first made its appearance. Establishing the validity of this relationship is particularly important when workmen's compensation is involved. A patient with chronic backache may have acute exacerbations, as do many farmers in the spring and fall when their work load increases. An unremitting backache accompanied by fever, and loss of appetite and weight may lead one to suspect an infectious process or a metastatic tumor.

Thus, the physician should keep in mind infections, diseases such as the arthritides and osteoporosis, tumors—particularly ones that are metastatic to bone, in the older age groups—and mechanical causes such as acute and chronic strains, degeneration of discs with and without rupture, and congenital anomalies.

EXAMINATION

In the evaluation of any set of symptoms, including those associated with low back pain, it is essential that a system review and a general examination be included at some time in the course of the evaluation. This is important in that a patient's low back pain may well be due to disease metastatic from other organs. Without inquiring about such symptoms in other areas, or examining those sites, one can easily miss the cause of the patient's pain.

For the sake of expediency, however, only the examination of the back and lower extremities, as well as pertinent roentgenograms, will be considered here. Very little equipment is needed for an adequate examination of the back and extremities. A percussion hammer, a pin and a tape measure are useful, though not essential. A stethoscope is necessary for auscultation of vessels. Assuming that the patient hasn't an acute low back pain that is too painful to permit his standing up, the examination is started with the patient in a standing position. His posture is observed for any evidence of antalgic flexion, or lateral lumbar list or "sciatic scoliosis" as a result of protective muscle spasm, which could be due to muscular, ligamentous or disc disturbance, including herniation. The direction of the list is noted. Since excessive lordosis may produce low back symptoms, the degree of lordosis should be noted. Flattening of the lumbar spine may indicate a rheumatoid spondylitis. Atrophy of the gluteus maximus can be ascertained by inspection and also by palpation as the patient pinches the glutei.

The patient's gait is observed to determine whether a limp is present, or whether the patient has an antalgic gait toward one side or the other. He is then asked to hop on one foot and then on

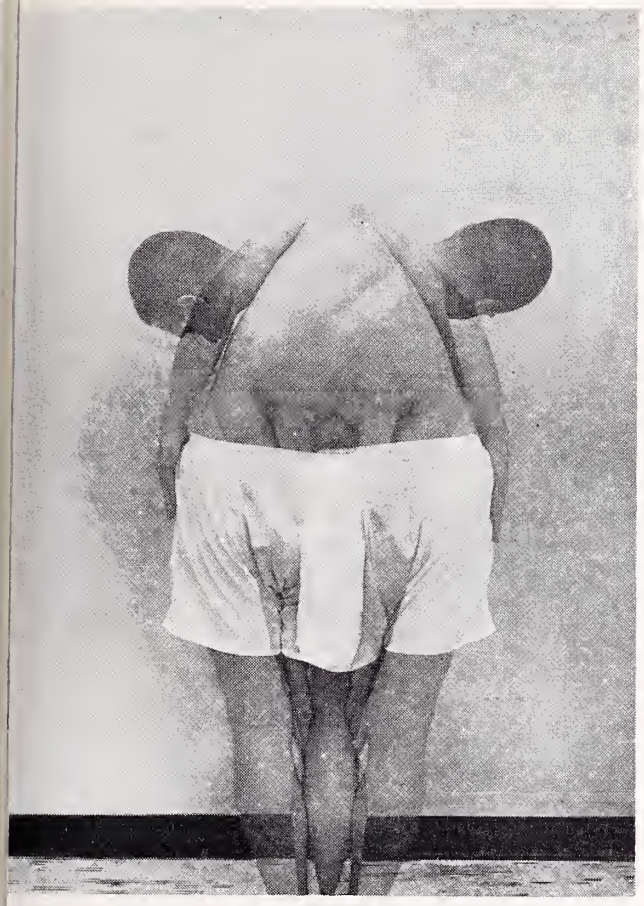

Figure 3. Lateral bending of the spine.

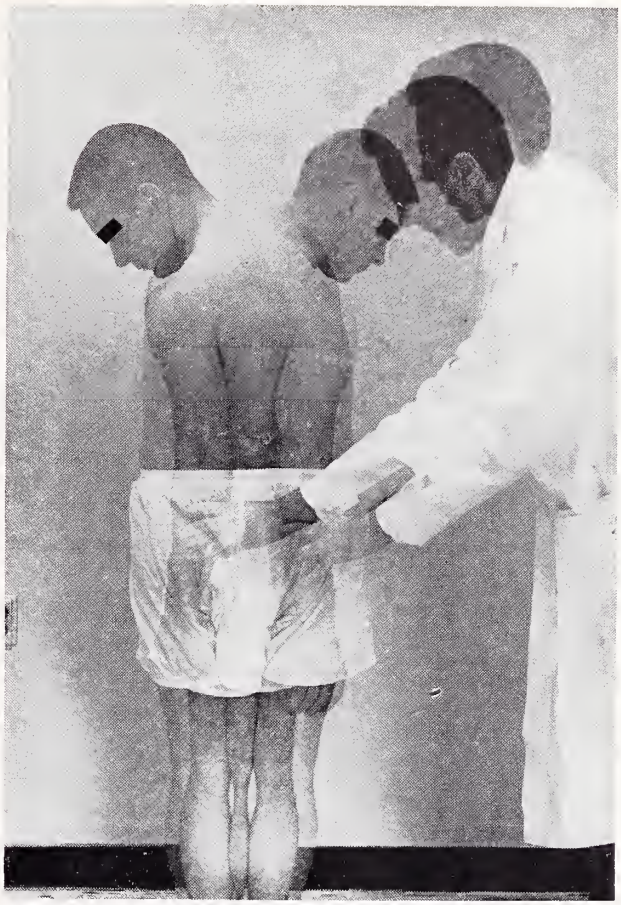

Figure 4. Rotation of the spine.

the other so that the physician may determine whether there is any weakness in the leg muscles, or whether pain is reproduced in the low back region. Such compression pain might well indicate disc herniation or degenerative disc disease, with instability.

While the patient is standing, motions of the back are tested. From a neutral position, most patients are able to flex forward from 70° to 90°, although with increasing age this may decrease (Figure 2). Extension is then tested. If symptoms are aggravated with extension, facet joint arthritis may be present. Lateral bending and rotation of the spine are also tested (Figures 3 and 4). Pain may be reproduced on one side or the other by these motions, or sciatica may be aggravated. This test will assist in localizing any disc herniation that may be present, or any localized facet degeneration.

The patient is then asked to lie on his back on a firm table or bed, and is made as comfortable as possible. Comfort may require flexing the knees slightly or using a pillow under the patient's head and shoulders. At this point, the straight-leg raising test is carried out, and the degrees of straight-leg raising are noted (Figure 5). If pain or restriction is present, the location of the pain is ascertained—particularly contralateral radiation of pain, which is indicative of nerve-root compression. In addition, dorsiflexion of the foot at a point just a few degrees less than maximum straight-leg raising is asked for as a means of determining whether there is aggravation of the pain. Another means of applying this same test is gradually to extend the flexed knee, with a finger pressing upon the tibial nerve in the popliteal space. An irritated nerve root in the lower lumbar spine will be revealed by this maneuver.

Figure 5. Straight-leg raising test.

Following the straight-leg raising tests, a Patrick's test is done (Figure 6). Its purpose is to indicate whether any hip-joint involvement is contributing to the patient's symptoms. At times, the differentiation between a sciatica with low back pain, and symptoms due to hip joint disease may be quite difficult. A negative Patrick's sign usually means a normal hip, or at least an asymptomatic hip.

A test for sacro-iliac joint involvement may be carried out at this time, though it has been my experience that the sacro-iliac is rarely involved in the low back problem. The test involves flexing one hip in order to fix the pelvis while the opposite extremity is hyperextended. Then the procedure is reversed. The result is the so-called Gaenslen's sign.

Reflexes can next be tested at the knee and ankle, and in the cremasteric area in males, with the use of the fingers or a reflex hammer (Figure 7). By slightly flexing the knees over my left forearm, I can test each knee jerk, and with the knees flexed about 70°, I can test each ankle jerk. Then the strengths of the dorsiflexors of each foot and ankle are tested, including the tibialis anticus, the extensor hallicus longus and the common toe extensors (Figure 8). Weakness of the extensor hallicus longus localizes the level of nerve-root compression, usually at the L-5, S-1 disc level. The foot evertors and invertors, as well as the plantar flexors, are also tested. It is important to test motor power with the knees flexed, in order to avoid any reproduction of the straight-leg effect on lumbo-sacral nerve roots.

In this same position, the calf and thigh can be measured for atrophy at the same distance from a fixed bony point. Similarly, sensation in the lower extremities can be tested by light touch with the finger or a piece of cotton. Testing with a pin may be necessary in the accurate outlining of an area of sensory impairment (Figure 10). This particular part of the examination may take considerable time in patients who have bizarre neurologic patterns or stocking glove configurations

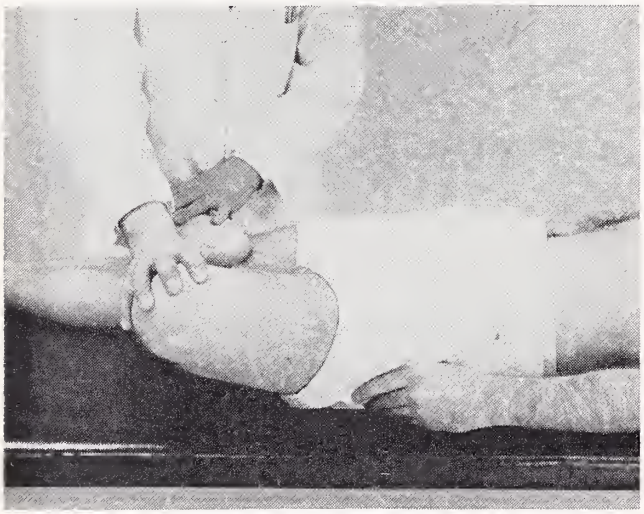

Figure 6. Patrick's test.

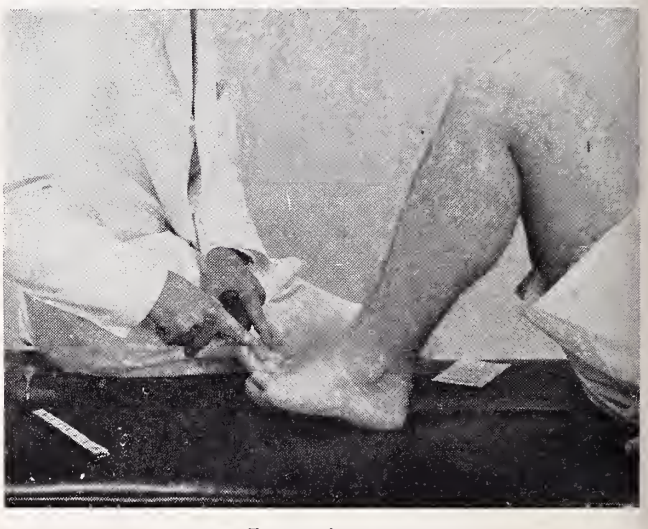

Figure 8. Testing for motor power.

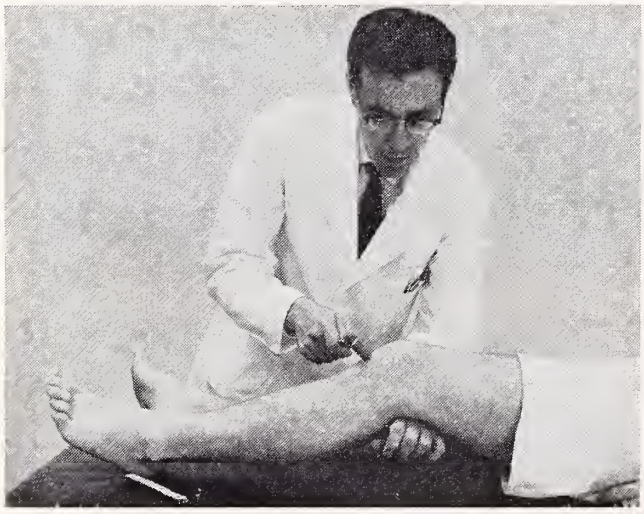

Figure 7. Testing for reflexes.

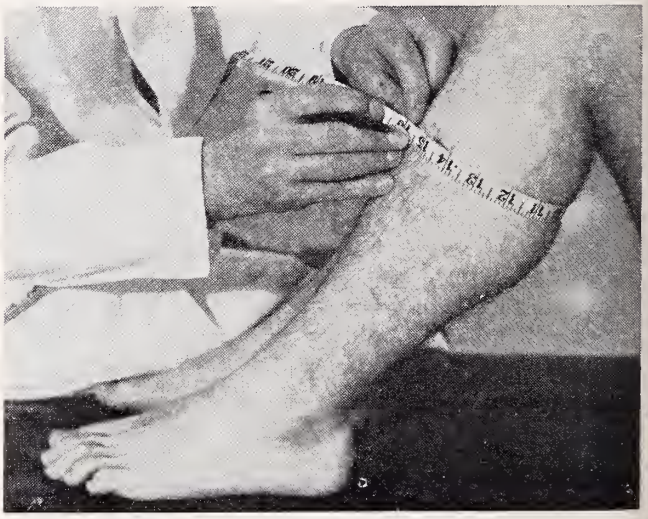

Figure 9. Measurement of calf for atrophy.

that may indicate psychoneurosis rather than an anatomic impairment.

In patients who are middle-aged or older, one should particularly examine the pulses in the lower extremities for the occasional instance of vascular occlusion in the abdominal aorta or in the bifurcation of the aorta, with symptoms of back pain. With the patient still supine, the jugular compression test and the abdominal compression tests are done to determine whether there is any effect of intraspinal fluid block. Palpation of the abdomen for presence of a bruit is done at this time.

The patient is then asked to turn to the prone position. If necessary, a pillow is placed under his hips to help him assume a comfortable position. One should note the method he uses in turning himself, for the amount of his guarding the low back region during the process of turning is frequently an indication of the severity of his problem. One should observe at this point whether the erector spinae muscles continue in spasm when the patient has reached the prone position, or whether a deformity which had been observed in the standing position has disappeared. It isn't uncommon for a lumbar list to relax and disappear as the erector spinae muscles relax in the prone position. Again, gluteal muscle atrophy can be looked for. Palpation of the spinous processes and iliac crests is carried out.

Areas of tenderness are noted, as well as areas of deformity or depression of spinous processes. One might note a step between the fifth lumbar spinous process and the first sacral, indicating a spondylolisthesis. Pressure on each spinous process from the upper thoracic region down is applied with the heel of the hand, with the patient in as relaxed a position as possible (Figure 11). This type of pressure amounts to the transmission of passive motion through the interspinous ligaments, the facet joints, the posterior longitudinal ligament and the intervertebral disc. If pain is present rather superficially, there may be kissing spines with a bursa between the spinous tips, or particularly if multiple tender spinous processes are present, the patient may be hyperreactive. If the pain appears on the deepest pressure, there may be involvement of either the facet or the disc unit. One should also note whether the pain is referred by this maneuver.

Those areas that have proved tender to this passive compression are then tested with the patient actively extending his back (Figure 12). Compression with the patient actively extending his back may aggravate the symptoms, and if so, one suspects that involvement of the articular facets or an extruded disc fragment is at the seat of the trouble. The tenderness may be relieved, in which case it is likely that instability of the disc unit is present. If the pain and tenderness remain the same, the test is indeterminate.

The clinical findings and the results of the test for instability should be correlated with roentgenographic findings, particularly with the flexion and

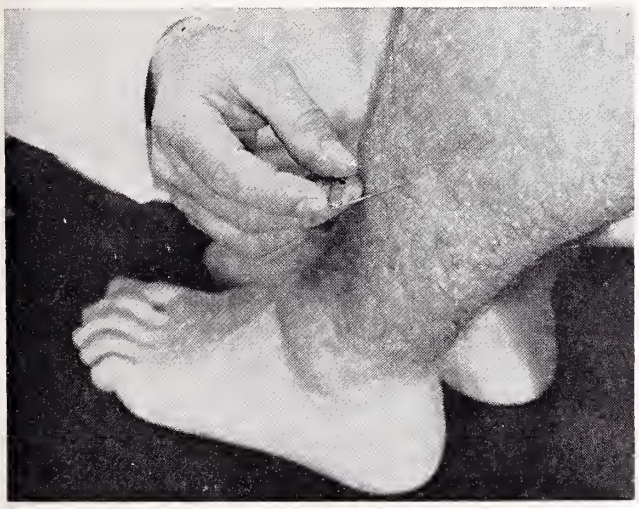

Figure 10. Testing for sensory impairment.

Figure 11. Instability test. Passive hyperextension.

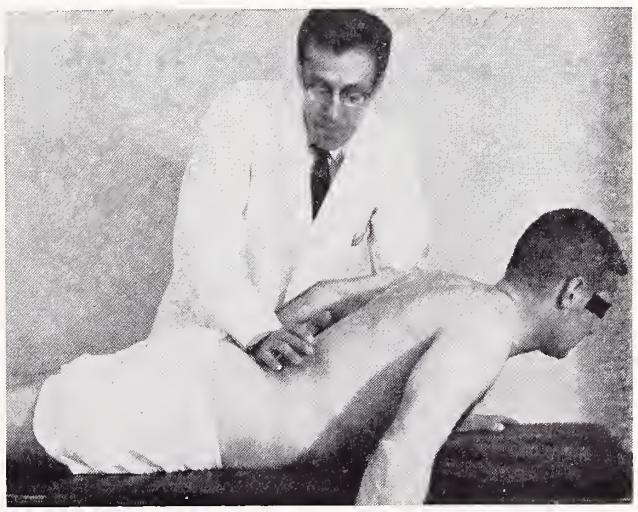

Figure 12. Instability test. Active hyperextension.

extension lateral views of the lumbosacral spine.

As the last part of the examination of the back, pressure is applied in the sciatic notch to ascertain whether any pain is present in that region. If there has been some sciatic radiation, usually the involved side will be tender.

Roentgenograms of the lumbosacral spine are an essential part of an evaluation of a patient with low back pain, although they often only corroborate the clinical findings. The most important views are the anteroposterior and flexion and extension laterals. In addition, oblique views, both right and left, may be obtained so that one can look for defects in the facet joints or in the pars interarticularis or the laminal arch as associated with spondylolysis and spondylolisthesis. The flexion and extension lateral roentgenograms are of value in determining whether excessive motion is present at a given disc level. That finding tends to indicate degeneration at that disc and possible instability if the clinical findings include a positive instability test.

It is important to recognize certain minor congenital variations so as to avoid placing too much significance on them as one determines the cause of the patient's symptoms. Congenital or developmental variations in the lumbosacral region are common. Probably five per cent of the population have them. As a rule, their presence is only incidental. A spina bifida occulta only rarely gives rise to symptoms, and when present, the symptoms are neurological. An asymmetry of the articular facets—the so-called facet syndrome which produces familiar catches in the lumbo-sacral region—may give increasing difficulty and require a spinal fusion for definitive treatment. A sacralized fifth lumbar vertebra, either partial or complete, is itself rarely responsible for symptoms, but may be associated with degenerative disc disease at the level immediately above. Thus, the symptoms are due to disc degeneration. A lumbarized first sacral, either partial or complete, likewise is not of itself responsible for symptoms. Spondylolysis and spondylolisthesis may be present in varying degrees with or without symptoms. The symptoms are usually secondary to degenerative disc difficulties. Frequently conservative management will prove sufficient for the relief of symptoms.

A large body of the roentgenograms of patients with lumbosacral pain are normal. The great majority of findings are intervertebral disc changes such as narrowing of the disc shadow, and increased or abnormal motion in flexion and extension. These disc changes may be accompanied by marginal ossification of the vertebral body. Degenerative arthritis of the lumbosacral spine is not uncommonly observed. In addition to the disc changes, changes in the articular facet are present so that the whole articulating unit of disc and facets is involved, with marginal osteophytes and narrowing of the disc.

Many other conditions may give rise to low back pain, including infections, both pyogenic and granulomatous. As a rule, the history gives important clues to the presence of an infectious process. Similarly, tumors, both primary (benign and malignant) and those metastatic to the lumbar vertebrae, may be suspected prior to clinical and roentgenographic examinations of the back. One should keep in mind, however, that considerable destruction of a vertebral body must have taken place before roentgenographic evidence of it appears. Thus, a normal roentgenogram is not necessarily a certain indication that metastases are absent.

Since carcinoma of the prostate may metastasize to bone, a rectal examination is an essential part of the back examination in males. In females, similarly, a pelvic examination may reveal pelvic malignancy, with spread to adjacent vertebrae. To my knowledge, a retroverted uterus does not *per se* produce back pain.

CONSERVATIVE TREATMENT OF LOW BACK PAIN

Acute Pain With Muscle Spasm. The back may be taped as the patient lies in bed with a pillow under his hips, or as he stands, with his trunk slightly flexed at the hips.

Bed rest should be prescribed, on bed boards if the mattress is too soft, and a semi-flexed position of hips, knees and trunk is advisable as a means of flattening the low-back area. The patient should be instructed to turn with his trunk in a straight position and his knees and hips flexed. Bedrest will bring relief to herniated-disc patients after lengths of time ranging from one or two days to three weeks. Symptoms will resolve in a great majority of patients. Analgesics, sedation and heat may be used as needed. Traction is of value only as a means of keeping the patient in bed.

Subacute or Chronic Back Pain. (1) The patient should be given the following instructions on the proper use of his back: (a) Bend with knees flexed, especially when you intend lifting an object, either light or heavy. (Neither the patient nor anybody else should keep his knees straight when he bends over.) (b) Stand with one knee flexed and with the foot resting on a footstool, or the equivalent, to relieve or prevent strain on the low back joints. By this means, you can avoid strain during such activities as bending over a sink to wash dishes, clothes, face, etc., and it is useful when ironing, paring vegetables, standing at a work bench (or, for that matter, at an examining or operating table). (c) When you sit, you should choose a firm chair and place your thighs and buttocks well back in it. A small pillow or backrest will be useful, especially in an automobile. Avoid soft overstuffed chairs and sofas. (d) Lying on a firm but not too hard mattress with the knees flexed over a pillow is often helpful. Bed boards between the mattress and springs will help to make a soft bed firm.

2. Support for the back by means of a lumbosacral corset or a low back brace can aid the patient in maintaining proper posture and can remind him to avoid strain. The support is to be used while the patient is most active at his work.

3. Exercises to develop the back and, particularly, the abdominal muscles are essential as a means of establishing control of the lumbosacral spine. The patient lies on his back and, with his knees flexed and his feet hooked under a sofa, bed or cupboard toe-board, alternately raises himself to and lowers himself from a sitting position. First, he does the exercise 10 times while extending his arms to the front; next, he practices the exercise while holding his hands behind his head until he can do a series of 10 sit-ups twice daily. He can strengthen his back muscles by doing extension exercises, starting from the prone position, with one or two pillows under the hips. Hyperextension should be avoided, and 10 exercises twice a day will be sufficient. In some instances weights are put on the chest for additional resistance to the muscles.

The exercises should be delayed until the pain has subsided enough so that it won't be aggravated.

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Low Back Pain

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ST. LOUIS, MISSOURI

PAIN IN THE BACK has long been an affliction of the human race, and in all probability of other vertebrates as well. Like headache, backache may have any one of many causative factors, and in some patients, several causes may operate at the same time. Some of the more common causes of backache are listed in Table 1.

When one glances through that partial catalog, it becomes obvious that a very careful and detailed history and physical examination is necessary for each patient who presents himself with such complaints. Oftentimes it is possible to make a reasonably accurate diagnosis from the history and physical findings alone, and even in difficult cases it is usually possible to narrow the field of diagnostic possibilities so that appropriate further studies can be instigated. It is always advisable to attempt making as accurate a pathological diagnosis as possible early in the course of the patient's difficulty, and for this reason appropriate x-ray studies of the involved area should be obtained before the patient has suffered for any considerable period of time. However, our desire to establish an accurate pathological diagnosis should not compel us to use all of the possible diagnostic tools on every patient when first seen. The history and the physical and x-ray examinations should allow the physician sufficient insight into the average patient's problem to let him arrive at a provisional diagnosis and to permit the establishment of a regimen aimed at relief of the patient's symptoms.

Because space is limited, it will be impossible for me to discuss all of the various diagnostic

Dr. Reynolds is a professor of orthopedic surgery at Washington University, St. Louis, and he made this presentation on April 25, 1961, at the IMS annual meeting.

TABLE 1

COMMON CAUSES OF BACKACHE

Extra-Skeletal	
Angina	Pancreatitis
Aortic Aneurysm	Intestinal Obstruction
Mediastinal Inflammation or Tumor	Retroperitoneal Tumor
Pleurisy	Prostatitis
Gallbladder Disease	Kidney Disease, Including Stone
Gastric or Duodenal Ulcer	Female Pelvic Disorders
Neuritis—Polio	Infection
Hysteria	Tumor
Peripheral Vascular Disease With Arterial Insufficiency	Retro Position
Skeletal	
Postural	Spondylolisthesis
Short Leg	Fracture
Strains and Sprains	Degenerative
Myositis and Fasciitis	Scoliosis—Old
Weak Muscles, Post-Polio	Obesity
	Congenital Anomalies
	Fracture—Acute or Old
Tumor	
Primary Spinal Cord Tumor	Giant Cell Tumor
Metastatic	Osteoid Osteoma
Myeloma	Aneurysmal Bone Cyst
Chordoma	Hodgkin's Disease
Hemangioma	
Metabolic	
Osteoporosis	Ochronosis
Anemia	Hyperparathyroidism
Calcification of Disc	Paget's Disease
Gout	

Infectious	
Blood-borne Pyogenic	Thyroid
Brucellosis	Post-surgery
Tuberculosis	Rheumatoid Spondylitis
Degenerative	
Poor Mechanics	Way of Life
Obesity	Heavy Work
Poor Cartilage Structure	Weak Abdominal Muscles
Heredity	Excessive Spinal Curves

possibilities, but I want to take up some of the commoner and more troublesome ones of them.

OSTEOPOROSIS

Osteoporosis is, by definition, an imbalance in the factors responsible for bone destruction and new-bone formation, in which the anabolic factors are less active than the catabolic, and over a period of many years the patient suffers a decrease in total amount of bone. This condition is not uncommon in elderly people, it is commoner in women than in men, and it is frequently found in not-so-old but post-menopausal females. As the condition progresses, a certain stage is finally reached in which the bone is unable to withstand the stresses that are placed upon it, and this difficulty is telegraphed to the individual's mind in the form of pain.

The onset of symptoms may be sudden, with a collapse of one or more vertebrae, or may be gradual, with a gradual settling and giving-way of the vertebral bodies. There is rarely any radiation of pain to the extremities, but there almost always is restriction of spine motion, with muscle guarding and widespread tenderness without neurological changes. The roentgenogram reveals a decrease in bony volume.

Where one or more vertebral bodies have collapsed, metastatic tumors and multiple myeloma are pertinent considerations. Metastatic cancer is usually not too difficult to rule in or rule out, but where there is doubt, x-rays repeated after a few weeks will usually distinguish cancer from the collapse associated with osteoporosis. On the other hand, multiple myeloma not infrequently presents clinically with back pain and osteoporosis, and in the x-rays it is indistinguishable from the results of senility and post-menopausal changes. Determinations of serum proteins and electrophoretic patterns, a search for Bence-Jones proteins in the urine, and (when indicated) an examination of the sternal marrow must frequently be carried out in order to establish the condition as an osteoporosis.

Treatment of the very acute case may require bed rest for a few days. However, one must avoid prescribing prolonged bed rest, for inactivity compounds the senile osteoporosis by adding the osteoporosis of disuse, which further weakens the spine. In very acute, severely disabled patients, small doses of x-ray therapy may give sufficient relief from pain to allow the individual to walk with the help of a brace. In others, a brace together with stilbesterol and testosterone therapy may be adequate to relieve the symptoms. The theory behind the use of these drugs is that they enhance protein metabolism, thus stimulating the manufacture of matrix so that new bone formation may take place. However, I'm not sure just how the hormones help to relieve pain in these patients. I've not had an opportunity to see evidence of new bone formation, even in patients who have been on these drugs for long periods of time.

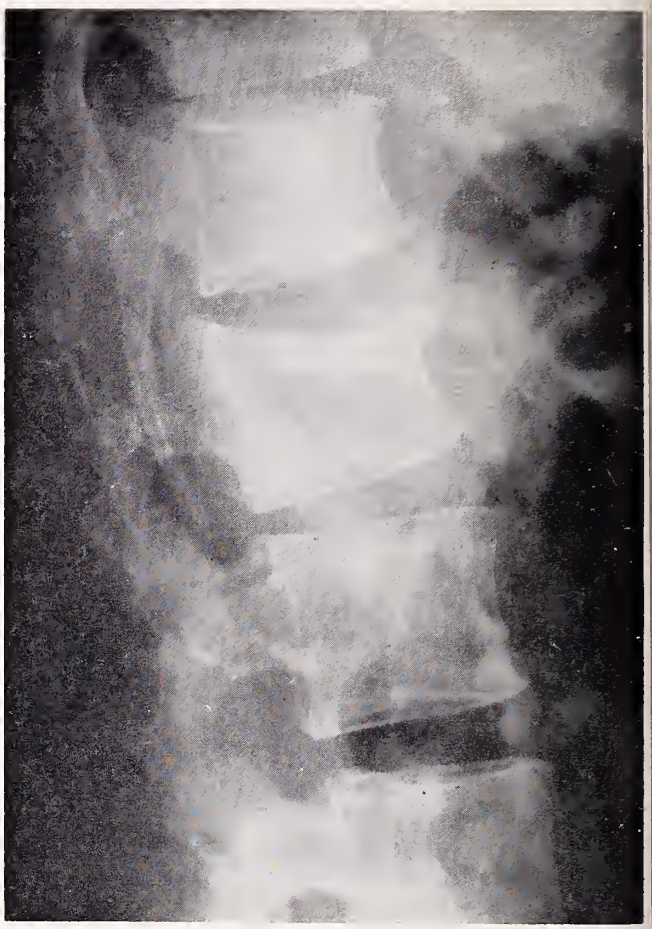

Figure 1. Compression fracture of the spine without damage to the posterior joints or ligaments. No reduction required.

As a rule, in patients who have osteoporosis, the laboratory test results are within normal limits. Yet, I have had occasion to see a few patients with severe bone alteration and severe pain that no form of treatment could relieve, and each of those individuals had a rapid sedimentation rate.

HYSTERIA

One of the most difficult if not the hardest differential diagnosis to make is to separate the patient with true low back pain of a structural or mechanical nature from the patient with a functional disturbance. Hysteria is quite common and must be constantly borne in mind by the physician.

At times, the history and physical findings will be adequate to make one suspect that a functional problem exists. At other times it is necessary to

follow the patient for quite a while before one can be reasonably certain that he is dealing with a functional disturbance. Psychiatric opinion and psychotherapy can be most helpful in the management of these patients. However, even the most experienced practitioner has been embarrassed at times to find that he has carried out a surgical procedure on a patient whose main difficulty was hysteria.

RHEUMATOID SPONDYLITIS

Rheumatoid spondylitis can be most difficult to separate from degenerative disc disease, and at times even from traumatic injuries. In the early stages of rheumatoid spondylitis there may be back pain, with or without sciatic radiation. There may be little or no stiffness, and no restriction of chest expansion. And although the roentgenograms will eventually show changes in the sacro-iliac and apophyseal joints, early in the process there may be insufficient change to warrant such a diagnosis. As a rule, the sedimentation rate will be elevated, and the patients are likely to complain of pain at night, and of having had to get up and sit in a chair to secure relief.

Repeated examinations and suspicion are of the utmost importance in establishing this diagnosis. In our series, despite our caution, we submitted at least one patient to an operation for ruptured intervertebral disc whose eventual diagnosis was rheumatoid spondylitis.

ARTERIAL INSUFFICIENCY DUE TO PERIPHERAL VASCULAR DISEASE

Arterial insufficiency due to peripheral vascular disease can produce low back pain or sciatica, or both, and it is present in an appreciable number of the patients who present themselves with those complaints. Careful interrogation of the patient concerning the sciatic pain will frequently uncover suggestions of intermittent claudication, if one keeps in mind that this phenomenon can occur in the hips and buttocks as well as in the calf. Searching for and determining the status of the peripheral pulses is essential in each patient. One must also remember that it is possible to have intermittent claudication producing these symptoms and still have palpable peripheral pulses. It is therefore important that the presence of a thrill or bruit be determined both by palpation and by auscultation. Further information can be obtained by auscularmetric examination.

FRACTURE

Low back pain and/or sciatica can result from a variety of injuries. There may be a compression fracture of one or more of the vertebral bodies. In these cases there is almost invariably a history of a type of injury that can cause hyperflexion of the spine, followed relatively soon by pain. If there is severe compression, there may be a prominence of the involved spinous process, and the area may be tender, but in many cases the physical examination fails to reveal any definite evidence of fracture. A roentgenogram, however, should confirm the diagnosis, although it may be impossible, on the basis of the x-ray, to estimate the length of time the compression has been present.

Acute compression fracture of a vertebral body without involvement of the posterior elements, or without evidence of damage to the interspinous ligaments, is a relatively mild injury. It is a stable fracture and doesn't require reduction and prolonged immobilization. Activity may contribute to an increase in the amount of compression, but the end results are far better when the patients are

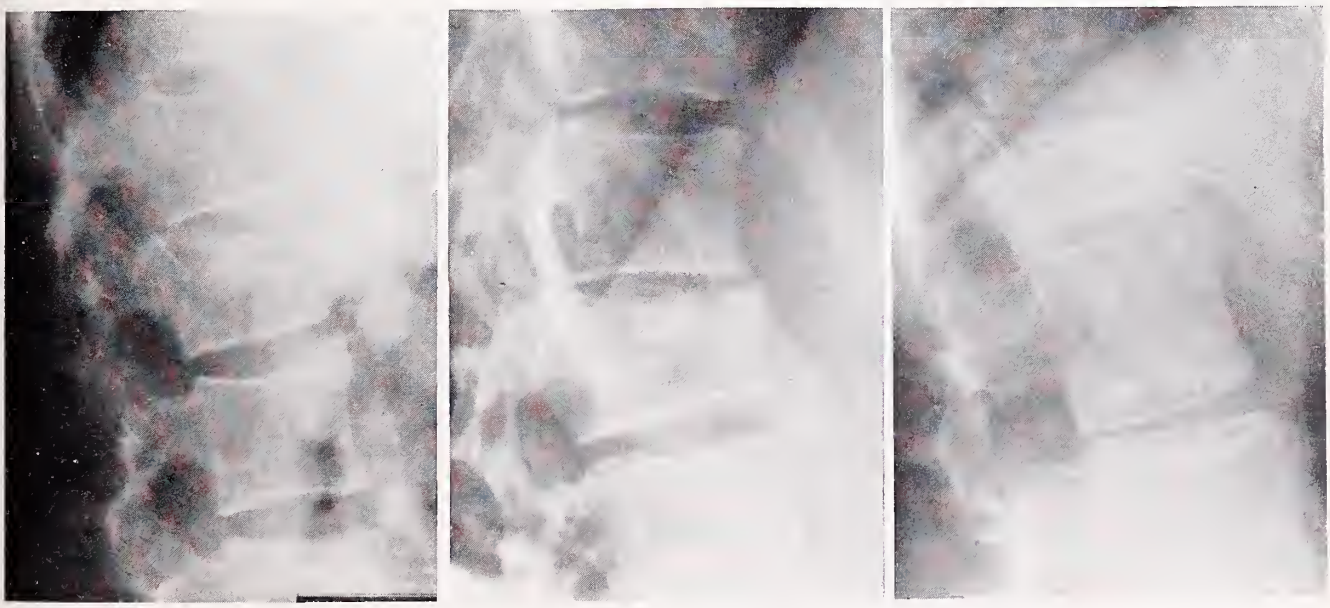

Figure 2. A. Lateral roentgenogram showing compression fracture, following closed reduction by hyperextension. B. Same case at the time of cast-removal. C. Appearance approximately one year following injury. Compression is essentially the same as originally seen.

permitted early ambulation and active exercise. If the fracture of the vertebral body is associated with damage to the posterior elements or the interspinous ligaments, or if there has been displacement, reduction and immobilization are of course essential.

SPONDYLOLISTHESIS

A history of recurrent episodes of rather severe low back pain, usually unassociated with much sciatica, in a relatively young patient who hasn't had any serious trauma, may lead one to suspect mechanical instability of the lower lumbar spine. A common cause of such instability is the condition known as spondylolisthesis.

By definition, spondylolisthesis is the forward displacement of one vertebral body upon another. This is made possible by anything that can separate the vertebral body from the posterior elements. Or the bone can remain intact, and yet advanced degenerative changes may produce so much wearing of the apophyseal joint that forward displacement occurs. In either of these circumstances, however, displacement cannot occur until the intervertebral disc has become so altered that it is no longer competent to stabilize the involved segment.

In patients in whom significant displacement of a vertebral body has occurred, it is possible to palpate the altered positions of the spinous processes. The spinous process and the posterior element of the involved segment remain behind, in normal relationship with the vertebrae and spinous processes that lie below, while the involved body and the spine above drift forward.

It is my opinion that all cases of spondylolisthesis resulting from interruption of the bony continuity are the results of fractures, whether from acute trauma or from fatigue. Since a definite familial pattern has been established a number of times, I think there must be some inherited characteristic that allows the bone to come apart more readily in the affected individuals than in the general population.

Many of these patients will require spinal fusion for relief of their symptoms. However, a trial of conservative therapy should always precede plans for surgery. When symptoms have developed in young individuals, the chances that they can be managed by the conservative regimen over a long period of time are not very good, and many of these will require operation. However, in the older individuals the chances of success with the conservative measures are much better, and not many of them are likely to require fusion.

I speak of spinal fusion intentionally, for I am unimpressed by the various procedures for achieving decompression of the nerve roots without fusion. In the vast majority of patients—even those having rather marked sciatica—a solid spinal fusion will relieve the back pain and the sciatica as

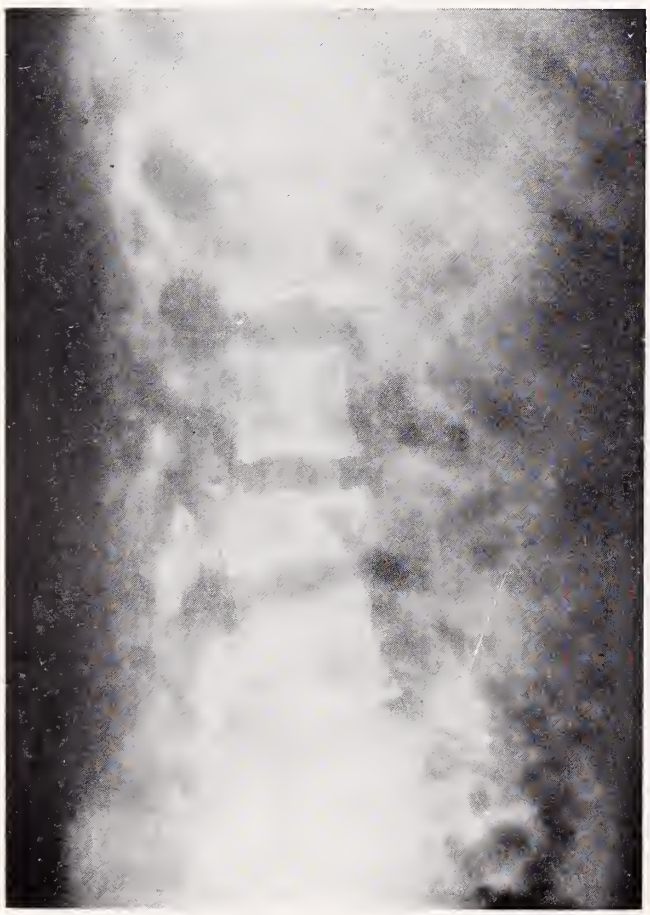

Figure 3. Extensive fractures involving the posterior structures as well as the vertebral body. Disruption of the disc and dislocation.

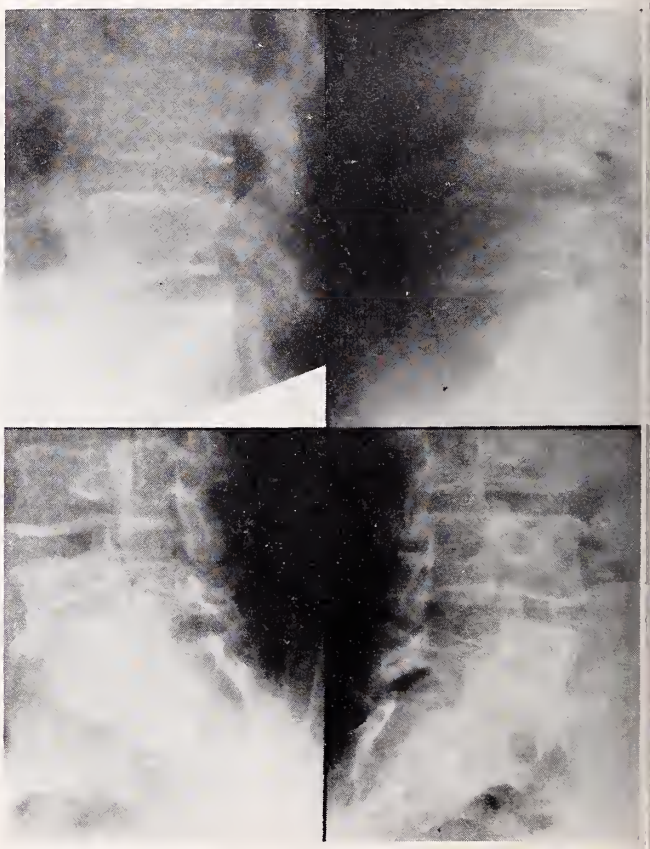

Figure 4. Pars-interarticularis defect without much displacement.

well. The incidence of ruptured intervertebral disc associated with spondylolisthesis is quite low. Yet, since there is a small possibility of it, those patients who have severe sciatica associated with spondylolisthesis—and in particular those with abnormal neurological changes—are probably most successfully managed by decompression of the roots on the involved side, together with a spinal fusion procedure.

SPRAINS AND STRAINS

By far the commonest cause of low back pain is acute muscle sprain or strain, resulting from a definite injury of the effort type, usually associated with lifting. Although the patient may be severely disabled for a short period, he is almost invariably relieved following a period of rest and locally-applied heat. In a survey of 600 such patients, I found that 96 per cent responded and were able to return to work within three to four weeks, almost irrespective of the treatment used, and the 4 per cent whose difficulties lasted for longer periods I found to have other conditions that the injuries had aggravated.

I shall have nothing to say under the heading *myositis* or *fasciitis,* for those terms mean nothing to me.

DEGENERATIVE CHANGES

Sooner or later, every spine will show x-ray evidence of degenerative changes. The pathological lesion is an alteration of the intervertebral disc, with subsequent decompensation of the disc. There are wide variations in the rates and in the manners in which the disc becomes incompetent in different individuals and in different races. A frequently referred-to etiological factor is that the spine was ill designed for the upright position, and that when man began to walk on his two legs, the altered body mechanics produced unusual and excessive strains on the lower lumbar spine. However, ruptured intervertebral discs are not unknown in animals, particularly dogs, and particularly in the chondrodysplastic varieties. Furthermore, museum specimens of various animals invariably reveal evidence of degenerative changes in the spine. One wonders, therefore, whether it isn't more logical to assume that the upright position has, in fact, served as a protective mechanism in man, and that rather than producing excessive strains, with early deterioration, it may not actually have retarded degenerative changes, thus making the spine more durable.

Williams has pointed out, and Lindbloom has

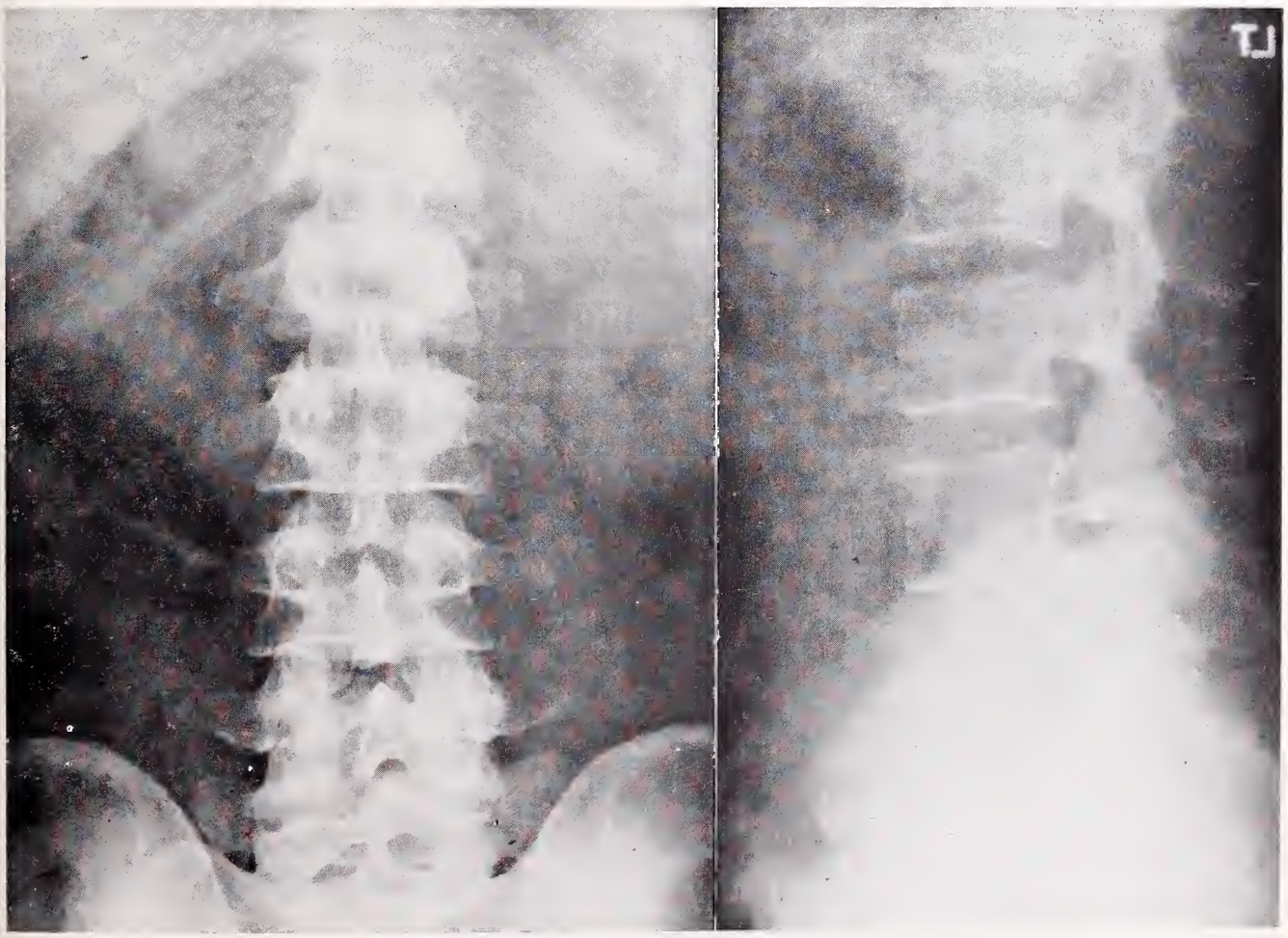

Figure 5. Spondylolisthesis of fourth lumbar vertebra on the fifth, due to degenerative changes in the apophyseal joints and the intervertebral discs. No bony defect.

more recently demonstrated in animals, that early degenerative changes occur in the spine on the concave side of any curve. It therefore should follow that the straighter the spine, the later would be the appearance of degenerative changes in it.

The intervertebral disc, from its earliest inception in the embryo and thence throughout life, is relatively devoid of circulation, and this fact may be important as regards the degenerative changes that occur in the nucleus pulposus and in the annulus fibrosus. Other factors perhaps even more important may be inheritance, the extent of spinal curves, the manner of living, the type of musculature, obesity, other metabolic disturbances, and the types of trauma to which the organism is subjected. The incidence of intervertebral-disc rupture in the white race is essentially the same in the male as in the female. We rarely see a ruptured disc in a Negro. Symptoms from degenerative disc disease occur more commonly in the third and fourth decades, and it is rare to see one in a teenager or in an individual past 60 years of age.

When degeneration of the disc unit occurs, it alters the mechanical function of the unit. As a result of this alteration in function, excessive stresses and strains are placed upon the apophyseal joints and on the supporting ligaments and muscles. These extra strains may in themselves produce low back pain, with or without sciatic radiation. These episodes tend to be recurrent, to have sudden onset and to be associated with some sudden although not unusual motion. And these episodes are usually relieved by rest, support and time.

In a certain percentage of these patients undergoing degenerative changes of the disc unit, the nucleus pulposus will herniate through the annulus fibrosus. This herniation may occur anteriorly, or to either side, or into an adjacent vertebral body. As a rule, when the herniation occurs into a vertebral body, little if any symptomatology is associated with the herniation. Such an event may be recorded roentgenologically as a Schmorl's node. When the herniation occurs to the side of the disc, there may be no symptoms other than those associated with altered function of the disc unit. At times, when the herniation occurs anteriorly, there may be some associated abdominal pain or reference of pain to the anterior aspect of the thighs, in addition to that associated with the malfunction of the disc unit. In other patients, the herniation of the nuclear material through the annulus fibrosus will occur posteriorly. When this happens, it is possible for this mass of fibrocartilaginous material to compress a nerve root, producing the syndrome of nerve-root compression. In this group of patients it is quite unusual to find evidence of back pain only. Much more commonly, there is back and leg pain. Others may have only leg pain. In almost every instance one will find some alteration of the neurological picture if he looks for it carefully.

Most patients with herniation of the nucleus pulposus, regardless of the direction or pathway it has

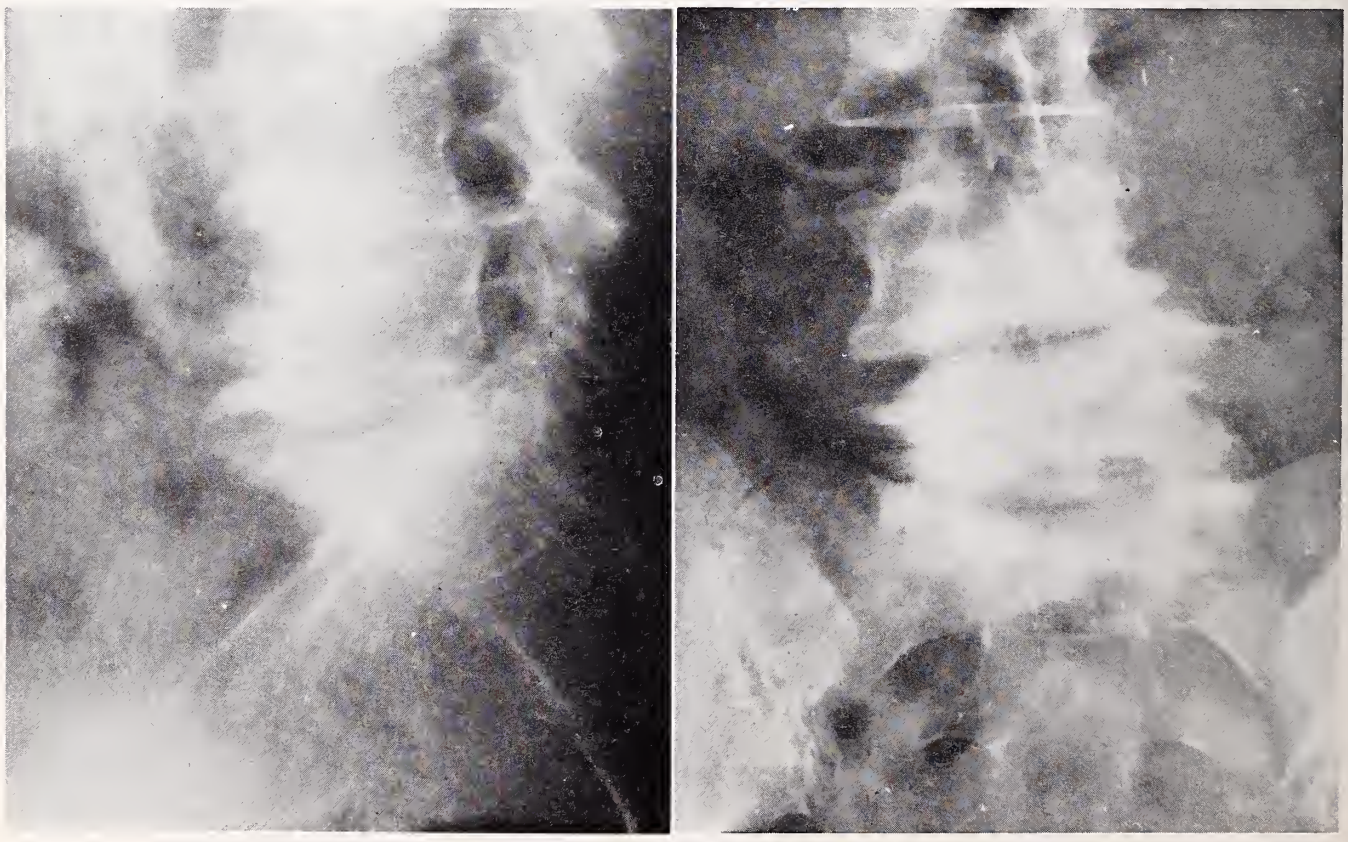

Figure 6. AP and lateral views of lower lumbar spine, showing advanced degenerative changes.

taken and regardless of whether there is nerve-root compression, will respond to conservative treatment. It consists of rest, support to the low back, and time. As time goes on, there is a tendency for this fibrocartilaginous mass to shrink up and to scar down, and this may be a factor in relieving some of the sciatica. In other individuals, as time goes on, the nerve root fatigues, and the pain may entirely disappear, though leaving considerable alterations in motor power, size and reflex response, as well as some areas of sensory alteration.

The response of nature, therefore, to a degenerative intervertebral disc and altered disc unit function is an attempt to stabilize this altered segment, with the production of inflammatory new bone along the ligamentous structure that spans the disc space from vertebral body to vertebral body. The results are the spurs that are visible in the x-ray and are frequently designated "osteoarthritis." In time, many of these spurs join, and the damaged segment is sealed over with bone. In other individuals, the altered segment is stabilized to a certain extent by scar, so that as the patients get older, they tend to have less trouble, both because of the healing effect and also, undoubtedly, because the structural demands are lessened.

Conservative treatment, then, consists of a low back support. In my experience, the most comfortable one is a corset with two heavy metal staves in the back, with solid back and side straps, and without a pad. Metal braces are difficult for women to wear because of the clothing that they prefer, and men have trouble too, for the braces wear out their trousers. The support does not immobilize the spine, but merely provides some restriction and reminds the patient to be careful. In addition, rest and local heat are helpful. Deep heat, either by short wave diathermy or ultra sound, is no more effective than that which is provided by a heating pad. Exercises to strengthen the abdominal muscles should be commenced as soon as the pain will allow, and should be followed by extension and flexion exercises for the spinal muscles.

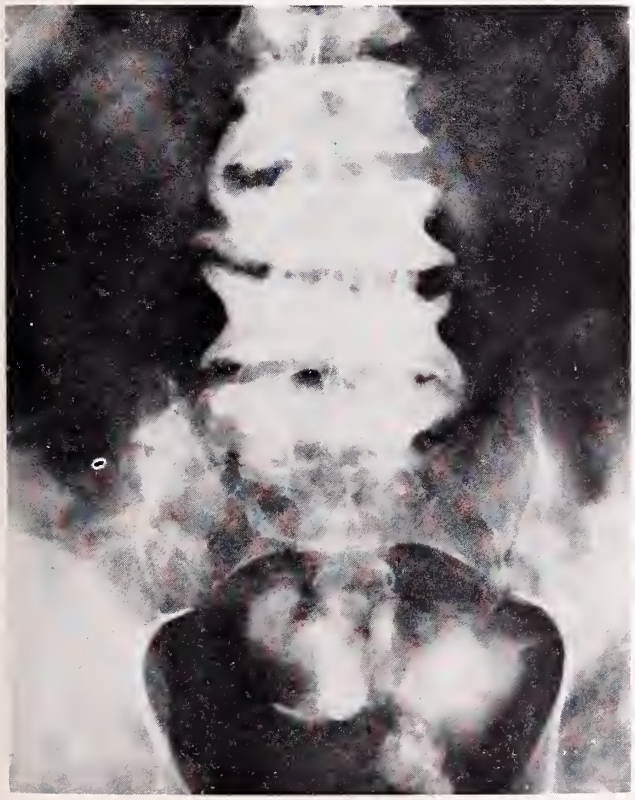

Figure 7. Another illustration of advanced degenerative changes.

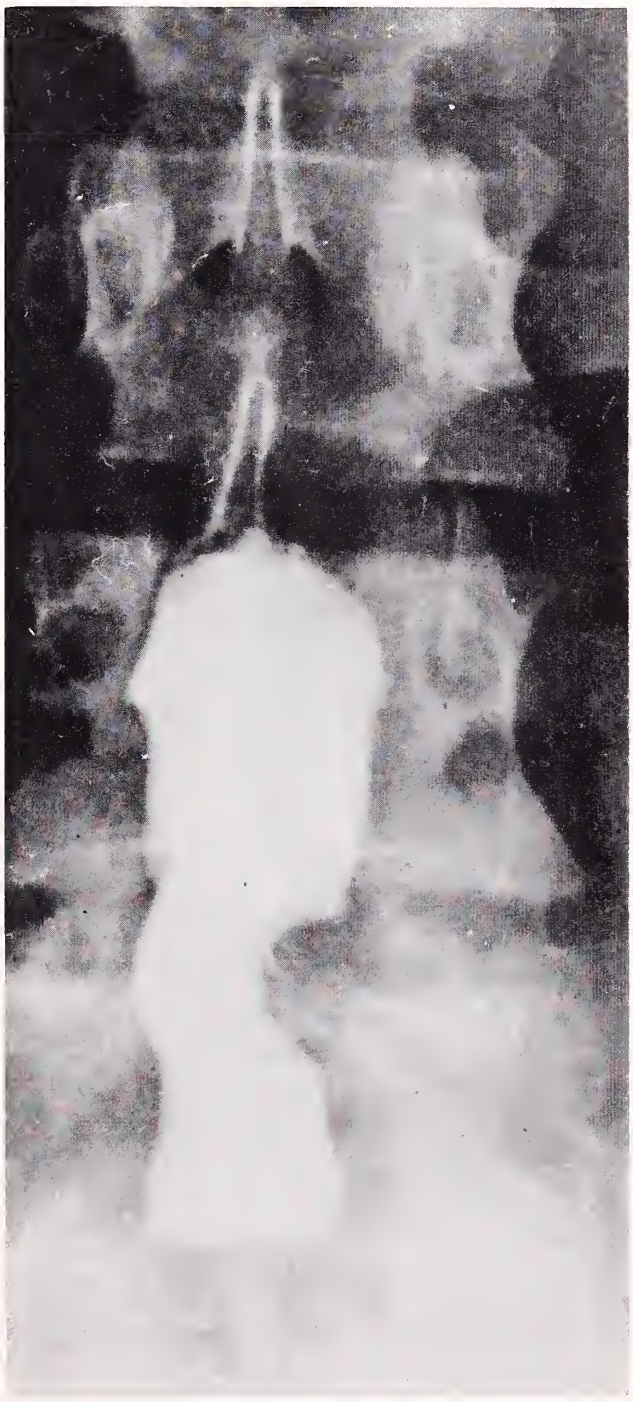

Figure 8. Myelogram showing lateral indentation defect.

A certain percentage of patients, however, don't respond to conservative treatment and require surgical intervention. Surgery is indicated in patients who show an acute picture of nerve-root compression, who show advancing or increasing neurological deficit in spite of absolute bed rest, and who have failed to respond to conservative measures within a reasonable length of time. Depending upon the demands placed upon the patient and his pain-tolerance, this period of time is usually somewhere in the neighborhood of three months.

Patients who fall into this group and are candidates for surgery should have some diagnostic localizing procedure prior to surgical intervention. This should be either a myelogram or a discogram. Although the discogram, when properly done, provides more information than the myelogram, there is always the possibility that an asymptomatic disc may be sufficiently traumatized by the insertion of the needle so that it will cause symptoms later. For that reason, we prefer the myelogram. It should be understood that a myelogram is not an entirely innocuous procedure, but since somewhere between 5 and 10 per cent of patients who have intervertebral disc rupture with nerve root compression have more than a single involved disc, it seems advisable to carry out such a procedure before deciding upon surgery.

Thus we come to the question of surgery. If we realize that the main difficulty with the patient is a degeneration of the intervertebral disc, with altered function of the disc unit, which is made up of the nucleus pulposus, the annulus fibrosus and the adjacent vertebra, it is immediately apparent that an operative procedure that would remove the pressure from beneath the nerve root by partial excision of the disc can be expected to do that and nothing more. The altered function of the disc unit will remain following such a surgical procedure. The ideal operative procedure, then, is one that will remove the pressure from the nerve root and will cause the two involved vertebral bodies to be joined together by solid bone. Although such a procedure, when successful, does place extra strains upon the movable segments above and below, it can be relied upon to produce healing of the most severely involved segment.

If these things be true, one asks why this is not the operative procedure in each case. The difficulty comes in trying to get bony union between the two vertebral bodies, both at the time of surgery and in the postoperative period. When the operation of spinal fusion, either by interbody fusion or fusion of the posterior elements, is added to the operation for removal of the disc, the magnitude of the procedure is more than doubled, with a resulting increase in the number of complications that are possible. In addition, some form of postoperative protection for the involved area must be provided until solid bony union has occurred, and this increases the period of post-

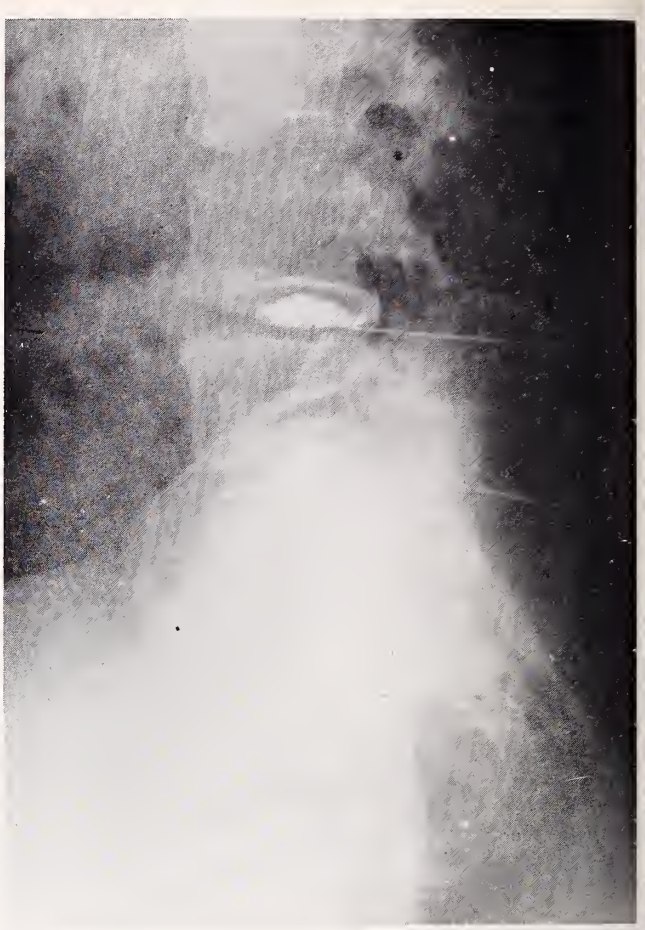

Figure 9. Discogram indicating escape of dye posteriorly at L4 and early degenerative changes at L5.

operative disability—usually about double that which is necessary following simple disc removal. And finally, about 20 per cent of the patients fail to obtain solid fusion, almost irrespective of the type of fusion performed.

Thus, in the survey of a large series of cases followed over a long period of time by several different observers, the overall end-results of fused as compared with unfused cases have shown about a 15 per cent better score for the fused cases. At the present time, and until someone can discover a better method of spinal fusion, it is my feeling that the treatment of nerve-root compression associated with intervertebral disc rupture requiring surgery should be simple disc excision. The results from this procedure will allow very satisfactory activities in 40 to 50 per cent of patients. An additional 25 per cent of the patients will be quite well satisfied to be able to carry on their regular activities, although they may have episodes, from time to time, of discomfort and pain that may necessitate the wearing of a corset or even bed rest for a few days. The remaining 25 per cent of the patients may have recurrent episodes of disability that will necessitate another operative procedure. For these last individuals, I feel that spinal fusion should be a part of the second operative procedure.

DRUGS

Throughout this entire discussion, you will note that I have said little about drugs or medications. By and large, none of them are worthwhile. The patient suffering from an acute muscle strain may obtain considerable relief from two or three days of the judicious use of steroids, but under no circumstance should steroids be used in any other situation, for they are ineffective even in rheumatoid spondylitis. Likewise, in my opinion the so-called muscle relaxant drugs are worthless. The most important medication is something that will relieve pain. However, it may be necessary to prescribe mild tranquilizers and vitamins in order to control the patient while he is waiting for time to heal the lesion.

Pathomechanics of Backache

CARROLL B. LARSON, M.D.

IOWA CITY

PHYSICIANS FOR generations have accepted the symptom of backache as being a natural consequence of diseases of the spine such as tuberculosis, osteomyelitis, spondylitis, metastatic disease and other recognized clinical syndromes. These are visible to gross inspection and give characteristic x-ray findings that are accepted as being related to backache. Laymen take for granted such other causes of backache as short leg, postural strains, excessive lifting and focal infections. In the absence of known diseases, such x-ray findings as asymmetric facets, spondylolysis, sacralization and vertebral-body spurs have been suspected of being causally related to backache. Architectural changes of one kind or another, when present, have come to be thought of as necessary to explain low back pain. Since this seems to be the case, it is the purpose of this discussion to summarize what is known of the pathomechanics of the spine, and what—if any—causal relationships with backache may exist.

BASIC MECHANICS OF THE SPINE

A concept of the basic mechanics of the spine is in order at this point. The spinal column can best be likened to a flexible pole, and it is this pole that provides us with the means of remaining upright. Were the spine a rigid, straight pole, it would be difficult for us to resist the forces that tend to tip the body. Thus, nature has provided curves in the column so that it can better resist bending forces. The curves are developed in orderly and regular sequence from the continuously forward-bent spine of the newborn. The first functional demand is a curve to allow the head to be held upward, the next is a change to allow the sitting position, and later when standing is attempted, the pelvis inclines forward for balance. This last modification is likely to affect all of the previously established curves by tending to flatten and broaden them.

The result of these functional adaptations is that characteristic physiological curves persist in the spinal column, namely, cervical lordosis, lumbar lordosis and pelvis inclination. For the spinal column to remain upright, it must be balanced over the body's center of gravity. The muscles of the spine and abdomen act by proprioceptive control to balance the flexible spinal column over the line of gravity in the sagittal plane. There is great physiological latitude in the relative lengths and degrees of spinal curves, yet each curve is compensatory to its neighbor, with the result that the line of gravity, as it passes upward from the supporting base, intersects with all the curves of the spine at the determinable levels. Consequently, the body weight is more or less evenly distributed to the front and back of the line of gravity.

The relationship that exists between the curves and the line of gravity determines posture in so far as mechanics are concerned. The amount of lordosis present is, to my knowledge, dependent upon two factors. One is the inherent length of ligamentous structure between vertebral segments, and the second is the anteroposterior positioning of the acetabulum relative to the pelvis. In 1953, we investigated the latter by a special x-ray technic, and found a positive correlation showing that the more posteriorly the acetabulum is placed, relative to the anteroposterior plane of the pelvis, the greater is the lordosis.

NATURE AND FUNCTION OF THE DISC

A spine devoid of muscles and ligaments, except for the discs and their investing tissues, remains in the upright position and resists all efforts to bend it beyond its normal flexibility (Figure 1). The mechanism that allows this intrinsic equilibrium should be understood, for the disturbance of this equilibrium is the meat of this discussion.

Since the spinal column is a series of bony

Dr. Larson is professor and head of orthopedic surgery at the S.U.I. College of Medicine. He made this presentation on April 25, 1961, as the Erskine Memorial Lecture at the IMS annual meeting.

vertebral bodies fixed, one to another, by intervening discs, it would suffice to describe one level as representative of all, except for a few anatomic differences. A unit is made up of contiguous vertebral bodies with an intervening disc, and each unit maintains stability and flexibility independently of other units.

It is important to know the anatomic and physiologic properties of the disc on which rest this responsibility and the ability of the spine to distribute and absorb all gravitational and translatory stresses that are brought to bear on it. In observing the mechanical behavior of the lumbar discs, Hirsch has stated that in healthy discs, neither hemilaminectomy nor total laminectomy leads to any appreciable disturbances in the disc's behavior. Of all the soft tissues, then, the intervertebral disc carries the greatest responsibility for the preservation of the flexibility and stability of the spinal column.

The center of motion lies toward the posterior portion of the disc, and motion between vertebrae is associated with adaptational changes within the disc. The nucleus is semi-gelatinous and offers resistance to compression, since it is confined within the space outlined by the annulus at the periphery and the cartilaginous plates above and below. Since the nucleus normally contains 80 per cent water, it resists compression much as does any confined liquid. Keyes and Compere's description of the disc, written in 1932, is still substantially accurate: "The incompressibility of this portion of the intervertebral disc lends to it the function of transmitting the static and muscular forces from one vertebral body to the next, and establishes it as the axis of motion on which each vertebral body must move."

Changes in shape will occur under compression, and these changes depend upon the elasticity and stress-resistance of the confining ligament, the annulus. The diversity of directions of the fibers within the annulus indicates that it resists stresses in all directions. In 1935, Joplin described a point of weakness posteriorly, where there is lack of restraint from the bony rim, and also fewer and thinner concentric folds of fibrous tissue compris-

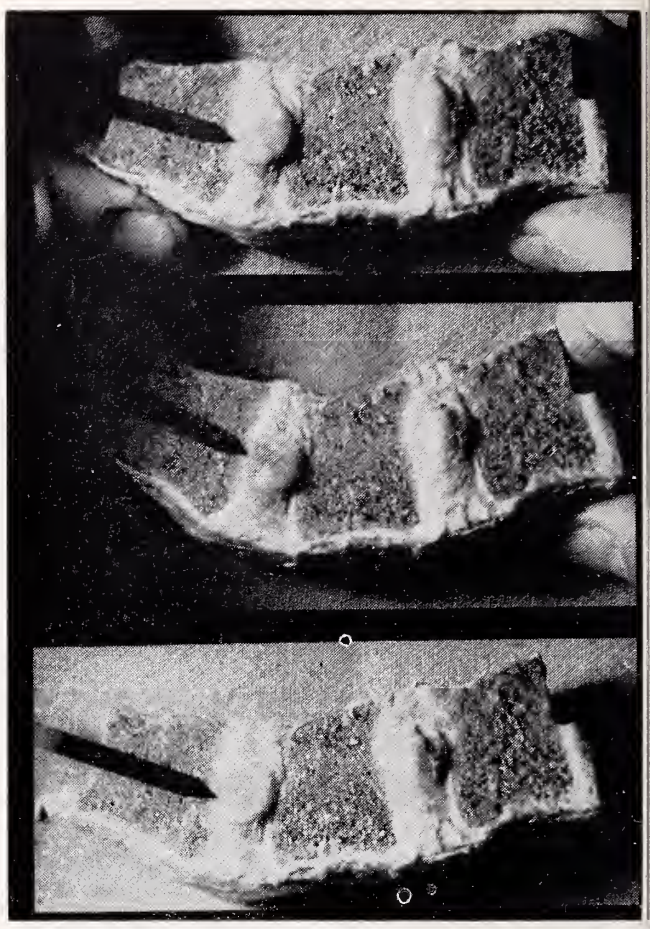

Figure 1. Section of split spine to show: (bottom) bulging of nucleus pulposus; (center) extension position, with compression of posterior annulus; (top) flexion position—annulus under stretch.

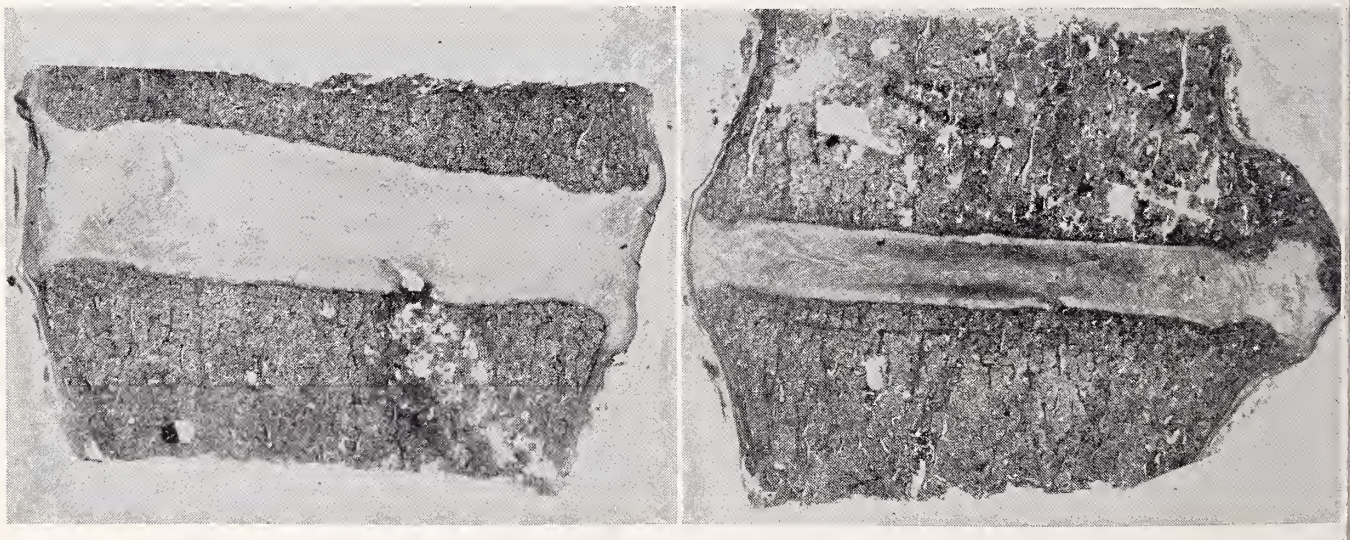

Figure 2. (A) Disc in late middle life. Cavity in central zone. (B) Disc in late life. Narrowing and osteophyte formation, increased fibrosis.

ing the annulus (Figure 2). The facets function as directors or guides to the motions that occur at each intervertebral level. Because of differences in form and position of facets at various levels, the directions of motion must vary accordingly. The facets in the cervical spine allow forward and backward motion in the sagittal plane, sideways in the frontal plane and rotatory in the transverse planes. In the lumbar region, the facets glide as parts of the surface of a cylinder, thus providing axial rotation, flexion and extension, but very little side motion. The side motion is taken up by the fifth lumbar segment, for the facets there are arranged more in the frontal plane.

Petter, in 1933, showed that a very definite expansion of the intervertebral disc occurs upon its removal from the body, and that further expansion occurs when the annulus fibrosus is sectioned. The average increase in height is 1.08 mm., and an average of 30.2 lbs. of pressure is required to reduce that expansion. These findings indicate that the normal disc maintains an intradiscal pressure greater than the forces transmitted in a state of rest. Any stresses of greater magnitude transmitted through the vertebral bodies must be resolved into two components: (1) the amount which is balanced by the intradiscal pressure and (2) the residue which is transmitted through, or absorbed by, the annulus.

Keyes and Compere, R. I. Harris, Inman and Saunders, Ghormley and others have studied postmortem specimens of discs from all age groups, and are generally agreed on the structure of the normal disc and on such structural changes as have occurred in each decade of aging. In infancy, the nucleus is well defined, and its line of demarcation from the annulus is distinctive. By the second decade, the transition from nucleus to annulus is much less definite. In the middle decades (Figure 2), the disc becomes more homogenous, and the fibrous elements become more definite. Cavities in the central zone of the disc are more frequent. Villus processes are evident, and there is more cellular debris as well as scattered nests of cartilage cells in the nucleus and more fibrocartilage in the subchondral region. In the later decades, the discs show dehydration and increasing fibrosis, often with some narrowing of the space.

Hirsch and Nachemson have studied the mechanical behavior of the disc and have concluded:

(1) If the disc is under load and has assumed an equilibrium, and then is subjected to an increasing strain for a short time, it resumes the form it had before the additional load was added (Figure 3). This power of adaptation to mechanical stresses, however, grows less as the load increases.

(2) By measuring expansile deformations of the disc under loads, they noted that the disc started

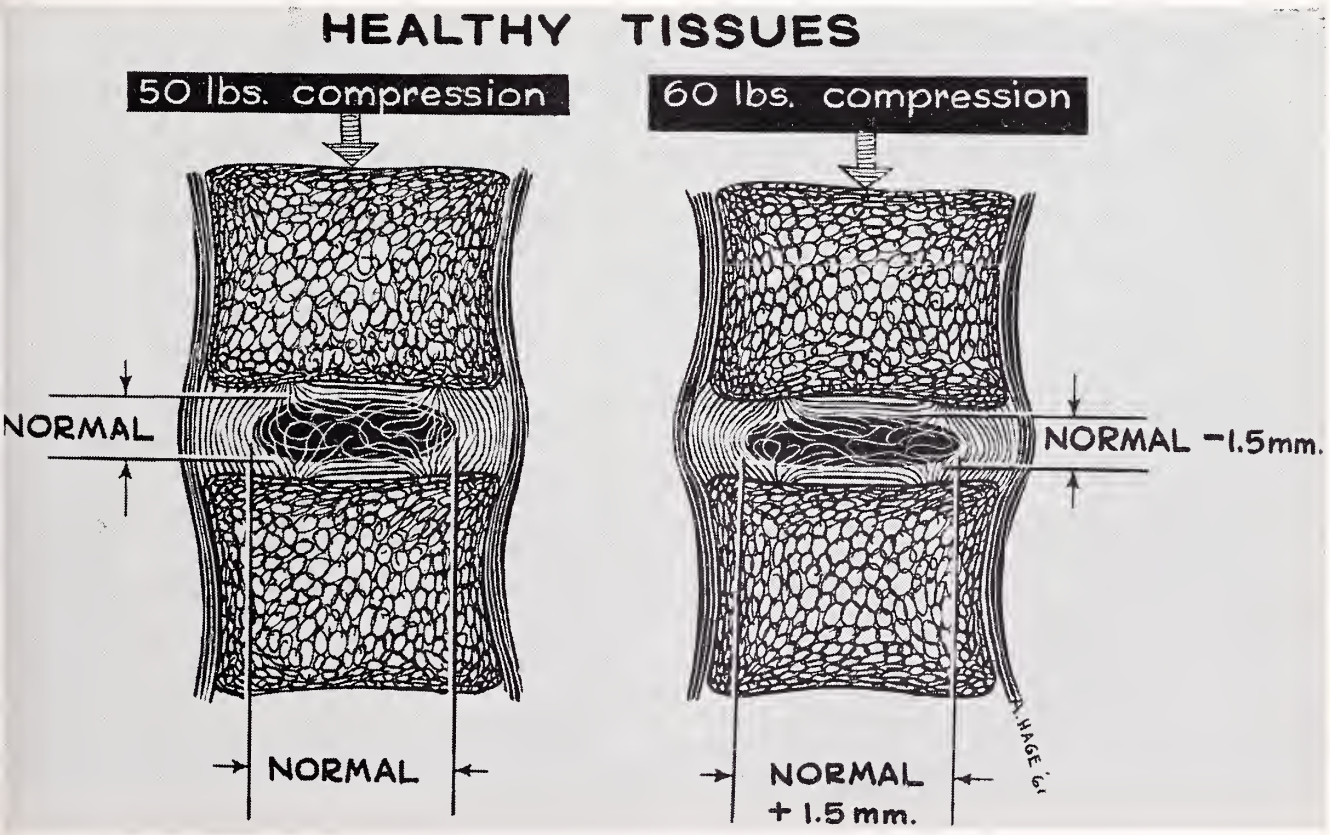

Figure 3. Schematic drawings to show effects of increased vertical loading on normal disc. The nucleus thins and broadens. The annulus resists nuclear expansion, but in so doing changes shape by increasing the bulge at the periphery. This is normal deformation.

to oscillate when subjected to rapidly acting forces.

(3) These oscillations have been registered while a disc was under a static vertical load of 10-130 Kg. This finding means that even if a disc has reached a static equilibrium, additional rapid forces can increase the deformation to a very great extent, even if the forces are relatively small.

(4) Compression of the normal disc may be repeated any number of times without disturbance in the mechanical response.

(5) The mechanical behavior of the disc differs according to the speed with which the stresses set in. If a disc is kept at a constant load, a certain amount of compression occurs until an equilibrium is reached. If subjected to a momentary load, it oscillates.

(6) It is striking to see how little the normal discs are deformed. The elasticity curves are the same shape as for any hyaline articular cartilage.

PATHOMECHANICS OF THE DISC

In the light of these observations, the mechanical behavior of the disc must be due to the biophysical construction of the disc material itself. Hence, to interpret mechanical dysfunction, we must consider the ability of the disc to hydrate itself.

The nucleus of the disc is composed of a three-dimensional collagen lattice containing a protein/polysaccharide gel. The gel is hygroscopic and tends to retain water. Although the collagen may of itself be hygroscopic, the water content of the disc diminishes with aging to parallel the diminished content of gel. Thus, it is probably the hygroscopic nature of the gel that is of most interest to us at this time. Furthermore, the water content must be held by a process other than osmotic pressure, since the smaller molecules which result from deterioration in aging should increase the osmotic pressure and hold more water. This does not occur.

Virgin, in 1951, suggested that imbibition, rather than osmotic pressure, accounted for the water-retaining property of the nucleus. Hendry, in 1958 and again in 1961, has shown that tissue from the nucleus propulsus, when placed in saline, will absorb considerable amounts of water. Nuclear material removed from backache patients likewise absorbed and retained normal amounts of water when unstressed, but didn't do so under the conditions that obtain in the intervertebral space. He deduced that the protein/polysaccharide gel was only moderately reduced in amount, but considerably reduced in efficiency in the backache patients. Far from generating an unusually high pressure, the nuclear tissue reacted to increased loads by losing abnormally large volumes of fluid, hence impairing the efficiency of the disc's hydrostatic mechanism.

Pieces of the annulus, in contrast, showed successive weight increases when submerged in

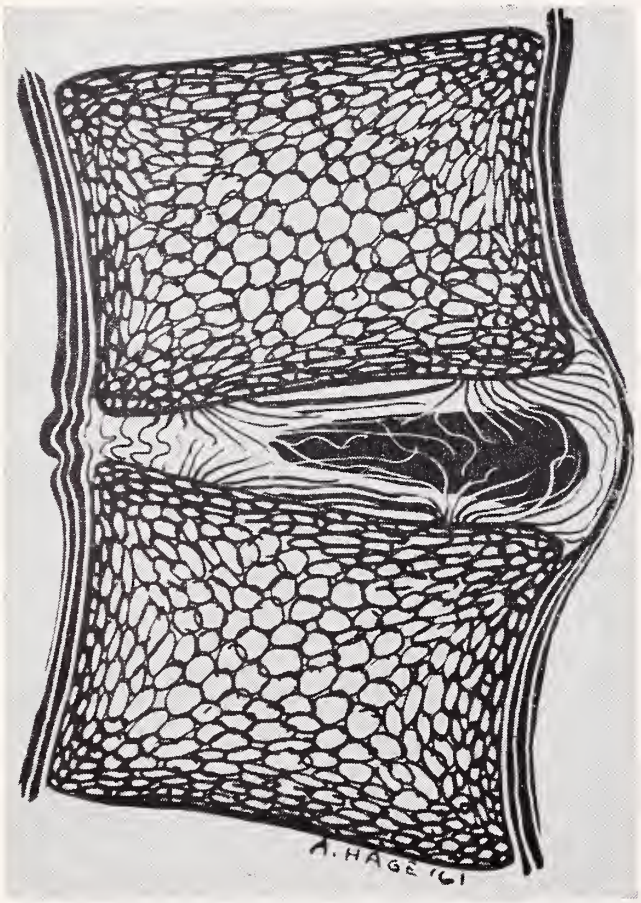

Figure 4. Schematic drawing, to show how the annulus undergoes compression in the deranged disc.

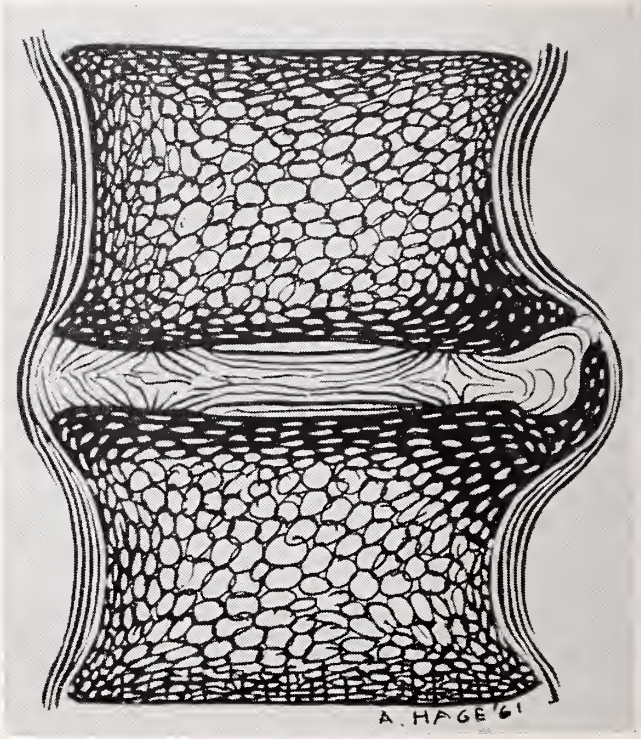

Figure 5. Schematic drawing, to show how fibrosis in the disc replaces the hydrostatic mechanism.

saline, thus following the laws of osmotically controlled hydration. We might summarize by saying that since normal intradiscal nuclear pressure, maintained by imbibition, is greater than the forces to be transmitted, the annulus is in a state of tension that increases with stress on the nucleus, but converts to compression on the annulus if the nucleus is deranged (Figure 4).

Since loss of gel and increase in collagen are part and parcel of disc aging, it is conceivable that each disc at some stage has an imbalance between the hydrostatic mechanism, allowing for instability and the collagen increase that tends to provide stability. In cases of backache, a cause or causes yet unknown may produce a loss of hydrostatic mechanism and have little to do with the aging process (Figure 5). Hendry has analyzed discs from cases of known backache, and has found changes indicating the content of hexosamine to be lower and the collagen content higher than those of aging discs, and those differences had no correlation with the age of the patients from whom the discs had been removed.

Whatever the etiology of the changes within the disc, it remains a fact that abnormal discs have a lower water content, and this in turn can alter the mechanical behavior of the disc.

As clinicians, our next problem is to translate our knowledge of the deteriorated disc into something more tangible that can be applied in the management of the backache patient. The fact that a disc is shown by x-ray to be narrow need not by any means indicate that it is the cause of the patient's pain, nor indeed need it ever have been symptomatic. The Munckfors investigation indicated that disc degenerations in themselves were not productive of symptoms until changes secondary to the degeneration occurred, such as herniation of the disc. Disc degeneration developed and attained advanced stages in some cases without corresponding occurrences of low back pain. Secondary changes seem to occur by chance and often without apparent reason, though such factors as infectious disease, dampness, drafts and certain forms of hard labor play provocative roles. On the other hand, static deformities such as pelvic tilt, lordosis, kyphosis and flatfootedness were of no significance. Lumbarization, sacralization and an asymmetric fifth lumbar vertebra did not seem to make individuals extraordinarily susceptible to low-back trouble. Spondylolisthesis was the single anatomic derangement that was accompanied by a high incidence of disc degeneration.

The aging disc which proceeds slowly and asymptomatically to narrowing and/or osteophyte formation must arrive at that point in a progressive and orderly fashion. The very slowness of the process allows the collagen increase to replace the loss of the hydrostatic mechanism. Hence, no excess of deformation occurs. Had excessive deformation occurred at any point, the likelihood is that the disc would have produced the symptom of pain.

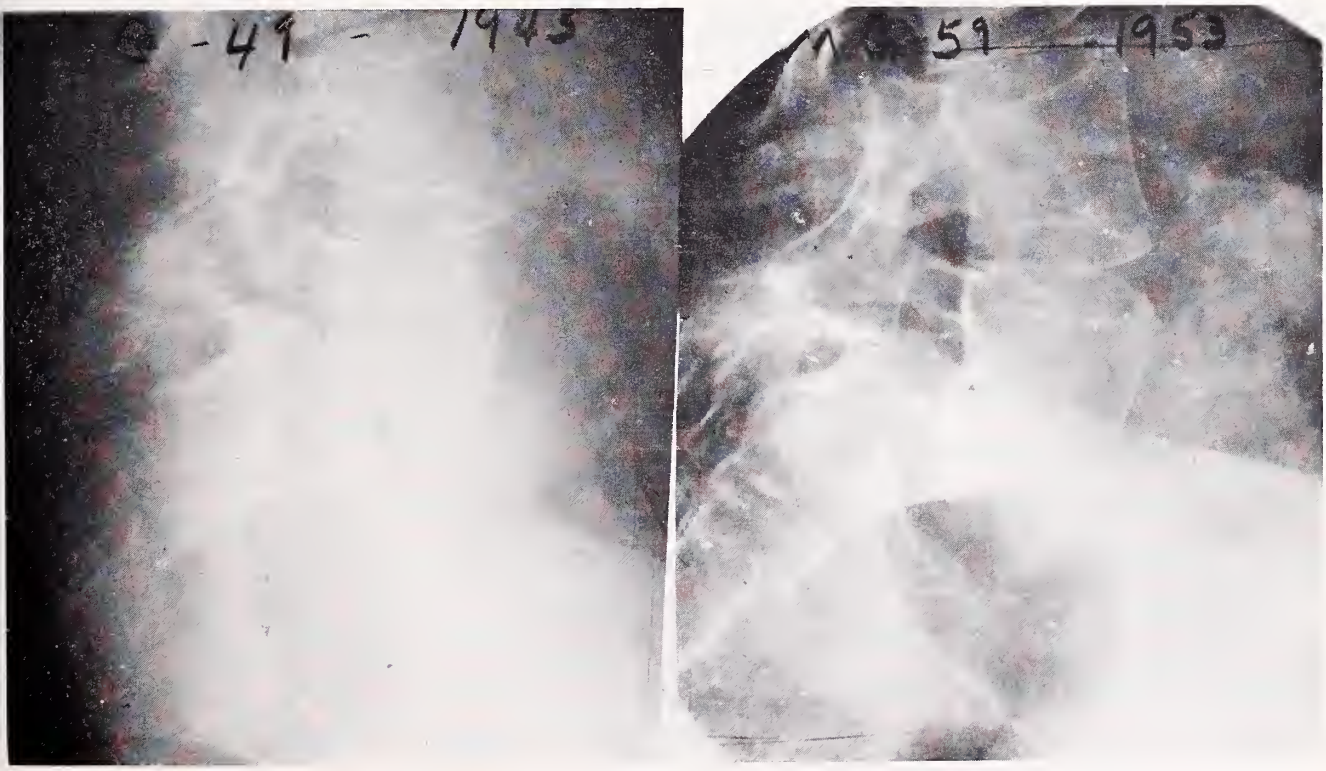

Figure 6. The x-ray on the left was taken when the spondylolisthesis was painless. The x-ray on the right shows the same case 10 years later, when the patient complained of pain. Note the narrowed disc at the spondylolisthetic level.

Thus we come to the question of the pain mechanisms implicated by disc changes. Wiberg studied back pain in relation to the nerve supply of the intervertebral disc, and was unable to demonstrate nerves in the nucleus or in the annulus. The ligamentous investment of the disc, however, is richly supplied with nerves, and these could be seen microscopically and tested clinically by palpation both before and after novocaine injection. It is well known to surgeons that the ligamentum flavum is non-sensitive, but the dura, the nerve roots and the posterior longitudinal ligaments are very sensitive, and any pressure over the disc itself is acutely painful. Novocaine in the disc space will relieve pain, and it must be assumed that the novocaine diffuses to the periphery of the annulus, where it meets the posterior longitudinal ligament, and that it blocks the nerve receptors there. This theory is verified by the fact that a blunt instrument can be placed in the disc space after the removal of a fragment, and pressure against the remaining disc tissue is not painful unless the peripheral areas of the annulus are pushed outward against the ligament with some force.

Roofe, in 1940, stated that the annulus and the posterior longitudinal ligament are innervated by fine, unmyelinated nerve fibers, the origin of which he was unable to determine, and he found naked nerve endings in the peripheral portion of the annulus, and both naked and glomerular terminations in the overlying ligaments. The nerve of Luschka has been said to carry fibers from the disc, but after two roots are anesthetized, external pressure on the disc remains painful. This somewhat rules out the likelihood of referred pain through the nerve of Luschka. Lindell had sectioned spinal nerve roots in cases of sciatica, and in 10 cases found that pathological changes were common, but he went on to say that in some instances at least, these changes could not be looked upon as the results of compression alone. He left the impression that a contiguous inflammation could be responsible for some of the changes. Suffice it to say that if these observations are correct, the likeliest site for the origin of pain in the disc would be the outer portion of the annulus and the investing longitudinal ligaments. To activate these nerve endings, there would have to be sufficient deformation of the periphery of the annulus and/or of the longitudinal ligaments, either by compression or by tension.

If the pain of disc disease is initiated by deformation, what are the circumstances and the types of deformation? I have already stated that deformation and recovery are not painful in the presence of normal ligaments and discs. Advanced changes in the aging disc have occurred without pain—hence, without deformation. Spinal fusion prevents deformation and thus relieves pain. Instability of the disc and/or rupture are painful. Thus deformation must have occurred, and it is particularly in relation to instability that I should like to go into some detail.

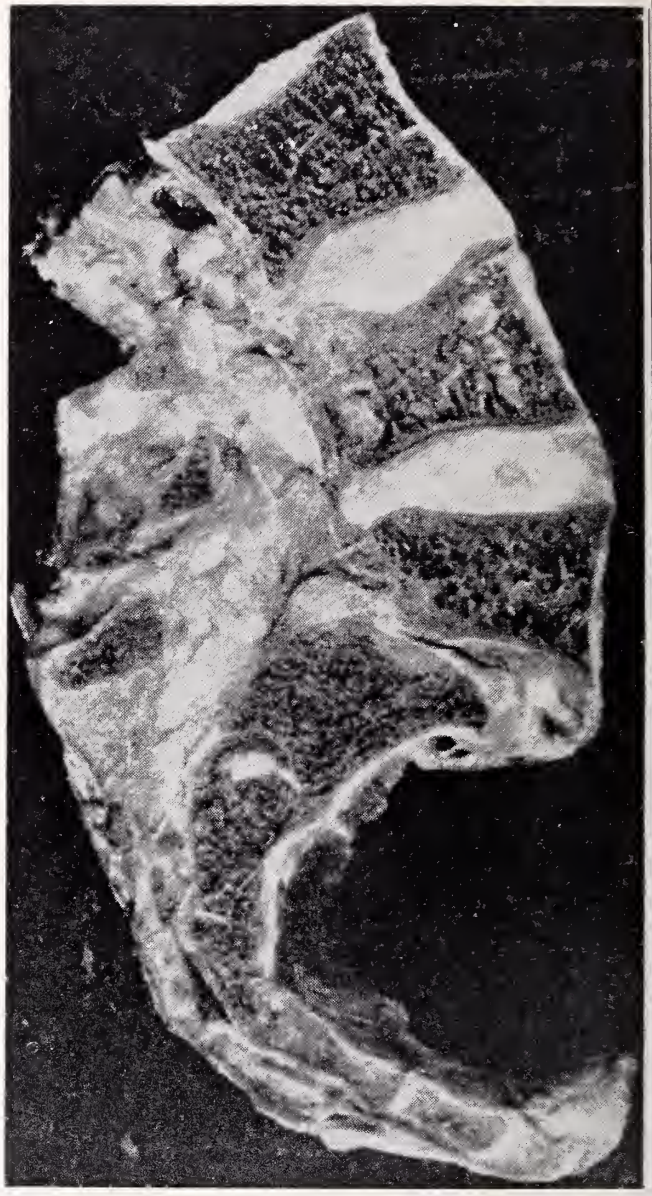

Figure 7. Postmortem specimen showing deteriorated disc at spondylolisthetic level.

SPONDYLOLISTHESIS

Spondylolisthesis is a well-recognized defect of the spine, and it is also well recognized that spondylolisthesis in many instances can exist without symptoms of backache throughout a lifetime. It is common knowledge, as well, that in many instances—sometimes rather early in life—backache can become a prominent symptom, and if we adhere to the hypothesis that the pain must be associated with deformation, it is obvious that the presence of spondylolisthesis, of itself, need not produce deformation. However, when the disc at the spondylolisthetic level deteriorates, it provides an instability that can produce deformation and

symptoms. As evidence in support of this hypothesis, I submit a case (Figures 6 and 7) in which x-rays were taken for reasons other than backache. The individual returned 10 years later with symptoms of backache, and the only detectable difference between the first x-rays and those taken on his second admission indicated that there had been a narrowing and an impairment of the stability of the disc at the spondylolisthetic level.

A schematic drawing (Figure 8) represents the possible shifts of one vertebra upon another where the ratio of intradiscal pressure to load pressure has gone up as the result of changes within the disc. In flexion and extension, deformations can stimulate the afferent nerve endings from either excessive stretch of the ligaments or excessive compression of what remains of the annulus. Knutsson, in 1944, described such excessive motion in the x-rays of patients with backache, and called it "the instability sign." Friberg and Hirsch, in 1950, reviewed 9,419 cases of backache and found that 39 per cent of them had x-ray signs of degeneration—meaning narrowing, osteophytes or both —and 50 per cent showed signs of vertebral-body shift. I hasten to point out that this radiological sign is somewhat difficult to interpret, and can be judged accurate only when the x-ray tube has been properly centered over the involved disc, and when the remaining discs above and below fail to show similar shifts.

A clinical test has been evolved in an attempt to corroborate the radiological signs of instability and to detect instability in discs that have not deteriorated sufficiently to present radiological evidence. This clinical test, which has been in use for 10 years and has been found helpful though by no means absolute, is accomplished by pressing downward upon the individual spinous tips, when the spine is in a relaxed, prone position. When this pressure produces pain, presumably as a result of deformation at the affected disc level, and when the pain disappears as active extension is added, the test is said to be positive. If pain persists when the spine has been actively extended, the test is equivocal, since the pain could arise from the facets that are in hyperextended position. We have insisted that the clinical sign and the x-ray sign must correspond before accepting instability as a diagnosis.

CORRECT AND INCORRECT MUSCLE STRENGTH RATIOS

So far, little has been said of outside forces that may act on the disc to cause deformations, and whether such forces can in any way be controlled. Empirically, it has been known for a long time that immobilization relieves many back pains, some of which undoubtedly would fit the criteria for disc instability. In lieu of immobilization, two potential factors have come to our attention. One of them is the status of muscles controlling spine equilibrium and spinal motion, and the second is the consideration of specific motions or spinal movements that are likely to exaggerate deformation in a deteriorated disc.

In 1955, we chose 45 subjects with backache and 19 students from the S.U.I. Postgraduate School to use as controls, primarily because they were available and were willing to enter a study. A special apparatus was constructed to test the strength of the abdominal and back extension muscles in a uniform way. Regulated and supervised exercise periods were then conducted, and after an average of 26 periods, the muscle strengths were reexamined. From the data obtained, it appeared that there is a normal ratio of back to abdominal strength, in the range of three to one. If this ratio is exceeded, the possibilities for backache are excellent, and reestablishment of the ratio through exercises produces a very high incidence of control of symptoms. Of the 45 subjects with backache, only two were unrelieved of their symptoms, and those two failed to obtain strength levels or ratios comparable to those of the controls. The remainder of the group showed a mean back-to-abdominal ratio of 2.88, which was slightly lower than the ratio of 3.2 for the control group.

An adequate explanation of the effect of muscle ratio upon deformation of the disc would be difficult, but it can be theorized that with proper muscle strength the fatigue factor allowing slouch positions should be diminished. Furthermore, it was oberved that the group was proud of its accomplishment, and this pride was reflected in better postural habits and better general muscle control of body movements.

The second external factor that has some effect on deformation is the way in which the total spine is used positionally, since the deteriorated

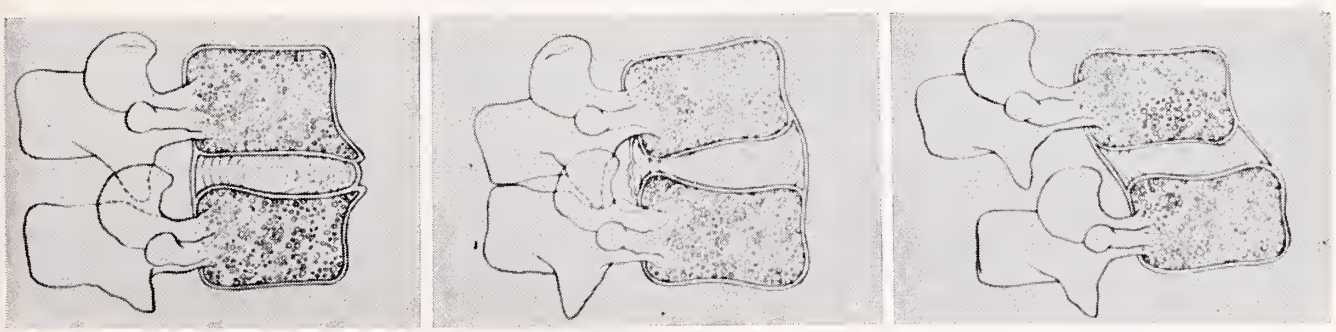

Figure 8. Schematic drawing to show possible deformations which can occur in a deranged disc under torque loading.

disc level can be thought of as a modified hinge. Usually at the fourth or fifth lumbar level, this hinge is placed where it has been estimated that a 120 Kg. man will carry a vertical load of approximately 40 Kg. If such an individual should bend forward through the use of his spine, keeping his hips and knees straight, the positioning alone would have the effect of converting the 40 Kg. vertical load to an increasing amount of torque as the forward angle increased. This torque, acting through an already deteriorated and perhaps unstable disc, would very likely cause deformation and consequent pain. This theory is borne out by the clinical findings almost universally, for stooping aggravates pain and will at times cause pain in an otherwise painless back.

Avoidance of this torque shift in disc loading is desirable, and if the patient complaining of back pain can be made to understand what he must do, and can be persuaded to apply his knowledge regularly in the actions of his daily life, the amount of pain created at the unstable level can be curtailed to a tremendous extent.

I might mention that braces applied to the low back are beneficial principally in this connection. First, the brace reminds the patient to avoid positions that enhance torque at the involved disc level, and second, it provides a proprioceptive fixed point against which the back can be maneuvered, as the patient either stands or sits, to allow for a more vertical loading of the disc.

CONCLUSION

In summary, then, backaches other than those caused by known disease may result from deterioration of a disc or discs. Deterioration allows for an alteration in the mechanics of the spine. The pain results from deformations that occur in the disc. Without knowledge of the etiology of disc deterioration, we have no specific therapy. Neither do we know the natural course of the deterioration, and thus can offer no prognosis.

Theoretically, any therapy aimed at prevention of deformation of the disc should at least partially control the symptom of pain. Practically, this is borne out in the use of braces, avoidance of torque strains in daily living, and spinal fusions. True, this treatment is empiric, but it does have foundation, and it is aimed at a resolution of the pathomechanics of the spine.

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Mercy Hospital Medical Day

The medical staff of Mercy Hospital, Des Moines, will present its Second Annual Medical Day on Saturday, October 28. The evening program at Hotel Fort Des Moines, at which Dr. Edward R. Annis will speak, will be jointly sponsored by the Polk County Medical Society and is to be open to the general public. Tickets for that session, including the social hour and banquet, are $5 per person. There will be no fee for the afternoon medical program.

SCIENTIFIC PROGRAM

Mercy Hospital Auditorium

1:35 p.m. "Newer Concepts Concerning the Etiology of Cancer"—John R. McDonald, M.D., Wayne University and Harper Hospital

2:05 "Recent Developments in Surgical Therapy of Cancer"—R. T. Tidrick, M.D., S.U.I.

2:45 "The Role of High Energy Radiotherapy in the Treatment of Cancer"—H. B. Latourette, M.D., S.U.I.

3:15 "Important Trends in Cancer Chemotherapy"—Sydney Kofman, M.D., University of Illinois

3:45 QUESTION AND ANSWER PERIOD

4:00 CLINICOPATHOLOGIC CONFERENCE—Dr. McDonald

6:30 SOCIAL HOUR AND BANQUET, Fort Des Moines Hotel
"Current Legislative Proposals in the Field of Medical Care"—Edward R. Annis, M.D.

The Pre-Employment Examination and the Low Back Syndrome

BILLIE H. SHEVICK, M.D.

MOLINE, ILLINOIS

PRE-EMPLOYMENT OR pre-placement physical examinations have been a part of the industrial scene for many years. There is no one presently at our company who can remember when they were not done. During the 50 years of their history, these examinations have gradually become more comprehensive. The emphasis has shifted to the total man. We industrial physicians now try to assess the applicant's total capabilities, and it is our aim to place each man in the job that he is best qualified, physically, to perform. The personnel specialists do have to take health and accident insurance, life insurance and retirement program eligibility into consideration, but we do hire people with all sorts of physical defects, and our rejection rate is low.

There is a definite need to develop pre-placement examination procedures that will provide us a reasonable evaluation of the present and future status of the low back. Throughout the nation, many industries have inaugurated programs to control the low back syndrome. Though predominantly a degenerative disease, this syndrome is regarded as an injury under the laws of many states, and it is in those areas that the need for control is most urgent.

A short abstract of an article entitled "Back Ache in Gynecology" that appeared in the March, 1961, issue of CURRENT MEDICAL DIGEST contained the statement that though low back pain is one of the commonest problems encountered in gynecologic practice, only 2 per cent of the cases are due to gynecologic conditions, the majority of them being due to orthopedic difficulties. Certainly those of us who practice occupational medicine would agree that the low back syndrome is a common entity, and that it is usually due to an orthopedic condition. Yet a special term has been coined to designate the low back syndrome in the industrial setting—*the industrial back*. The industrial back is no different from the low back syndrome in the farmer, the housewife or the businessman. There are some added psychiatric and social factors that change the patient's response to the disease, but the pathology is the same. The term may be partially justified by the fact that no other medical entity causes as much loss of time from work or is so expensive to industry as the "industrial back."

The word *problem* appears with remarkable frequency in the literature on this subject. All of us tend to refer to the low back syndrome as a problem. I agree, but the problem cases are relatively few. In more than 95 per cent of our cases, the response to conservative therapy is rapid. About 80 per cent of the patients are willing and able to do light work during the treatment period. These are not problem cases. Fewer than 5 per cent of cases present real problems to the medical profession and to industry.

Why do we say that these are problem cases? The problem is one of frustration. Frequently there is a complete lack of organic findings. Psychiatric factors are dominant, and they are difficult to distinguish from malingering. We can't cure the patient with a simple pill, although many products are promoted as curative. We don't have a simple laboratory test that will make the diagnosis for us. The patient is loathe to accept any discomfort, but refuses to change his way of life in order to live comfortably with the degenerative disease in his back. The results of treatment are poorer than our experience indicates they should be.

The problem is nationwide, and no one medical group has become famous for its good results in the surgical treatment of the industrial low back. In several reports, insurance companies have reviewed their results on a national basis and have concluded that surgical treatment for industrial back cases has been uniformly disappointing. Our orthopedic colleagues recognize this and freely admit it. It is standard procedure to report surgical results in two groups—the industrial group with poor results and the non-industrial group with good results. Some of this disparity reflects the fact that the evaluations were made before the final therapeutic maneuver, the application of the greenback poultice. But some of these cases will never be rehabilitated, even with a substantial financial settlement. It is part of our job as physicians to rehabilitate these people and promote their return to useful, productive work. We have a poor record as regards that part of our task.

Our colleagues in physical medicine frequently

Dr. Shevick, an industrial physician in the employ of Deere and Company, made this presentation on April 25, 1961, at the IMS annual meeting.

tell us that the difficult rehabilitation problems must be handled by specialists at the rehabilitation centers. A study was done at the Institute for Crippled and Disabled, in New York, covering 100 patients who had undergone rehabilitation following prolonged low back disability. Six of them had been returned to their former jobs "improved"; two had been placed in other jobs "improved"; four had been placed in other jobs but were still symptomatic; and 10 had entered retraining. Seventy-five per cent had been total failures with respect to rehabilitation efforts.

Two interesting points were brought out by that study: (1) The likelihood of recovery was much less if there were no organic findings (50 per cent of cases). (2) Only one in nine of the patients classified as having weak personality structure recovered, as contrasted with four out of five whose personality structures were classified as favorable. The patient whose low back pain is precipitated by a vague, slight motion, who has no organic findings, who fails to respond to any treatment, and who has a poor personality structure constitutes the hard core of the "industrial back" problem.

Aside from the medical problem, the difficulty for industry itself is simply one of high costs. In addition, there is the uneasiness of the corporation conscience—if you will—occasioned by a never-ending stream of back cases undergoing litigation. The corporation wants to take care of its responsibilities, just as does any responsible citizen. It is the duty of industry to develop health and safety programs that will prevent accidents and occupational diseases, and thereby prevent compensation claims. It is also the responsibility of industry to litigate and defeat unwarranted, exaggerated or fictitious claims. Industry wants to pay justified claims, and is not trying to avoid its genuine responsibilities. Rehabilitation is a basic tenet of its philosophy. The concept that the costs of injuries to workers should be borne by industry and not by the individual workman is commendable and well established. Yet, when costs continue to rise year after year, an attempt must, of course, be made to control them.

The costs associated with the low back sydrome are extreme. You are aware of the high medical expenses. In addition, the prolonged convalescence raises the weekly indemnity cost well above that for most other entities. Then, after comprehensive treatment and rehabilitation, we still have to make a settlement for permanent disability, if present, and that sum can be very high. In Iowa it can be as much as $23,500, which is just $500 less than double the benefit for accidental death. It is only a rare case in which the cost is that great, but the sums are, nevertheless, very high.

You are also well aware of the fact that these cases are numerous. They comprise between 25 and 50 per cent of the cases litigated before the Workmen's Compensation Commission in Iowa, depending upon the area involved. In other words, these comprise a substantial share of all the disputed cases.

These are the reasons why the practitioners of occupational medicine have been developing programs in an attempt to prevent or control the low back syndrome in industry.

THE DEERE AND COMPANY PROGRAM

The program that we are developing at Deere and Company is as follows:

1. We are selective in hiring new employees, and since we are in heavy industry, we will not hire poor risks. We try to place each man in a job that he is physically qualified to perform.

2. We still have a considerable number of accrued liabilities. Many of our present employees have chronic low back problems. We hope to develop a selective job placement program for our employees who have low back complaints.

3. The safety department in each plant is supervising a constant and sustained program designed to correct faulty work habits and contributing mechanical factors.

4. We have a training program for supervisors, since they have closest contact wtih the men who do the physically demanding work, and the success of our program must depend very largely upon the warning and teaching that they can do.

5. The medical department intends to provide comprehensive and sympathetic treatment for low back pain patients, but at the same time is determined to develop adequate records.

6. The decision as to whether a case is or is not compensable is made by plant personnel. We feel that the physician should not delve into the social and legal aspects of these cases, but rather should render a medical opinion based upon his own findings. In most of our plants, the personnel department makes these decisions with the aid of readily available legal counsel. The decision as to whether or not a given case is compensable will vary according to individual state laws.

The foregoing is a thumbnail sketch of what we hope to accomplish. Of course, one can't inaugurate such a program on one day and have it in full operation on the next. A sales campaign throughout the involved industry is required.

THE SCREENING OF APPLICANTS

I should now like to make some remarks about the selection of new employees. A pre-placement examination is given as a means of placing each man in a job that he is physically qualified to perform, but it is also intended to protect the firm against assuming ready-made medical liabilities. Thus, in a heavy industry such as ours, we attempt to screen out all candidates who can reasonably be expected to develop low back complaints in the future. Our grounds for predicting are inaccurate and unproved, and much work needs to be done before such forecasts can be made on an accurate and scientific basis.

If obtainable, the history is the single most valuable index that we have. When one evaluates the history and finds that the examinee has had recurrent episodes of back pain that radiated down the legs, or has had a single prolonged episode of disability for that reason, there is a reasonable certainty that he will have trouble in the future. On the other hand, however, a negative history provides absolutely no assurance that the examinee will have no low back trouble. In the Quad Cities area there are some industries that refuse to hire any applicant who has ever had any back pain, and other employers are placing more and more emphasis on back problems. As a result, applicants refuse to admit having had back problems, and I think it is fair to say that if you had children to feed and were seeking an industrial job in our community, you wouldn't admit having had bad back trouble, either.

Almost every applicant assures us that he has never had any trouble with his back, and we do no further questioning. We have attempted taking comprehensive histories, but have found them to be a waste of time. The examiner has to form his conclusions largely on the basis of his physical findings.

The physical examination is done with the patient stripped. We then observe his gait, his general physique, his muscular development, and his posture and coordination. In our examining room, the examinee walks from a booth to a scale, where he is weighed, and then to the area where he is examined. There is ample time and space for us to observe his movements. The examiner has a good conception of the physical requirements of the various jobs in the plant, and he tries to choose one that the applicant can do. If the candidate is small, with a slight physique and light weight, he is not given a heavy-labor job. Applicants who are either obese or extremely tall are suspect. I am especially suspicious of the tall man who has a disproportionately long lumbar area. One who has poor abdominal and gluteal musculature, with a lumbar lordosis, is a poor risk and should at least be singled out for more study. Scoliosis is observed, but static scoliosis hasn't been a problem for us.

During the course of the examination, forward bending is observed. It has been my experience that almost every applicant can bend forward adequately during a pre-placement examination. If the individual is a muscle bound type, with tight structures, and cannot quite touch the floor, I don't attach much significance to the finding, but there is a difference of opinion on this point. We routinely check the patellar reflexes, but we don't routinely check the achilles reflexes, look for sensory loss or measure the lower extremities for atrophy. If an abnormality is found during any part of the examination, we examine further. Previous back surgery will have left a visible scar, and this is a very important finding.

The predictive value of any part of the examination, as far as I am aware, is unknown, with the exception of previous back surgery. We have had enough experience with postsurgical cases to know that we are accepting an increased risk if we place such an applicant in a heavy labor job.

INCONCLUSIVENESS OF THE BACK X-RAY

There is no uniformity of opinion regarding the utility of pre-employment x-ray examinations of the lumbar spine. The experts are disagreed as to which abnormalities, on x-ray of the back, are significant in predicting that an individual will have back trouble. Only 40 per cent of the general population have normal spines, on the basis of x-ray findings, and thus the radiologists' concept of normality is, perhaps, questionable. We haven't done pre-employment back x-rays routinely, and I should like to report to you the results of the pre-employment x-ray program at Alco Products, in Schenectady, New York. In that plant, all employees both old and new were x-rayed, and the incidences of back injury in employees with and without preexisting defects were compared. There was no significant variation between the two groups. The Alco study revealed a few cases of opaque material in the neural canal and old compression fractures of the vertebrae that might otherwise have gone undetected.

The severities of back injuries, as reflected in loss of time from work, were as follows. There was no significant variation between the group with negative x-ray findings and the one with osteoarthritis and minor congenital deformities. There had been 50 per cent more lost time for the group with spondylolisthesis and pre-spondylolisthesis than for the negative group. The individuals with disc-space abnormalities had had twice as much, and those with prior surgical repair had had 13 times as much lost time as had had the negative group. Thus, the only findings of importance, according to the Alco study, were those having to do with spondylolisthesis and pre-spondylolisthesis, disc-space abnormalities and prior surgery.

Pre-employment back x-rays are not taken exclusively for their predictive value. In some states such as New York, which have a secondary-injury law that may apply to back cases, a knowledge of preexisting conditions is of value. It is entirely possible that the waiver provisions now in effect in Iowa may make the pre-employment x-ray examination more useful here. This is largely a legal question.

Some industries are rejecting between 20 and 45 per cent of applicants on the basis of the x-ray examination but, as I have said, we aren't doing pre-employment x-ray examinations of the back on a routine basis. Taking or not taking such films is left to the discretion of the examining doctor, and he bases his decision on the history and physical findings. He may be influenced some-

what by the knowledge of the type of work the man is going to do.

SUMMARY

The predictive value of a good history, properly evaluated, has been established, but the parts of the physical examination that provide reliable indications that an individual will have back trouble are, I think, unknown. There isn't even an area of general agreement. The ascertaining of facts in this respect is essential, and studies designed to gather them are, in my opinion, sorely needed. The experts disagree as to the abnormalities in the back x-ray that are of greatest importance in predicting the probability that the individual will have back trouble unless his work is limited. At present, the majority opinion seems to be that the important defects are spondylolisthesis, spondylolysis and narrowing of the disc spaces. The opinion of the examining physician, after he has marshalled all the facts and has evaluated them, is probably the best index that we have.

One of the important determinants of the final outcome in a back case is the personality structure of the patient. Disability due to chronic low back pain causes markedly varied emotional responses. If it were possible to employ only well adjusted people with favorable personality structures, the low back syndrome would not be the frustrating, expensive entity that it is. As I have said, the rehabilitation study conducted at the Institute for Crippled and Disabled in New York demonstrated that only one out of nine with weak personality structures succeeded in rehabilitation, as compared with 4 out of 5 with favorable personality characteristics. The personality of each patient was evaluated by means of a battery of personality tests, and psychiatric and psychological interviews. Favorable personality characteristics were diligence, composure, amiability, confidence, enthusiasm for work, motivation, cooperation and stability. What more could one ask for in a prospective employee? Such an evaluation, of course, cannot be made without comparatively long studies and personal interviews which make the evaluation too costly for ordinary employment practices at this time. An attitude and personality pre-placement study will be much commoner in the future than it is now.

The pre-placement physical examination is a tool that we have used many times in the past to control medical problems in industry. It has been effective in dealing with the compensation hernia problem, with the silicosis problem and with others. It is fundamental to the operation of any large industry. We naturally have taken this familiar tool and have used it in our attempts to deal with the low back problem. How good our results have been, we cannot know, for the measure of our success must be taken in the future. In this endeavor, we don't demand 100 per cent success. Any benefits we secure will be worthwhile.

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State University of Iowa College of Medicine

Clinical Pathologic Conference

SUMMARY OF CLINICAL FINDINGS

THE PATIENT, a nine-month-old male infant, was admitted to this hospital in the terminal phase of an acute illness. A two-year-old sibling was described as having had "bronchitis" with "coughing and wheezing" since birth.

The patient's birth had been an uncomplicated breech delivery, and his birth weight had been 7 lbs. 10 oz. Because of cyanosis at birth, he had received oxygen for three hours. Prior to going home with his mother on the third neonatal day, he had been examined and found to be a healthy neonate. During the following months, he had received "well baby care" that included the usual immunizations. He was a bottle-fed baby, and his growth and development had been within normal limits. No unusual health problems had preceded his terminal illness.

One month prior to admission, he had had the onset of a fever of 102°F. and of vomiting five to six times a day. That illness had lasted about two weeks and had been diagnosed as "flu."

After a few days of apparent health, he had had the onset of a 102°F. fever, of diarrhea with 10 to 11 loose, brown, foul-smelling stools, and of bronchitis. He was hospitalized for four days. No antibiotic therapy was given, and he responded to dietary therapy. During the following 10 days he received no therapy, and was considered to be healthy.

Six days before his admission at University Hospitals, he had again become ill, with the symptoms of a 103°F. fever, and of diarrhea with 20 to 30 stools per day. On his readmission to the local hospital, oral feeding had been continued, although some vomiting occurred. Four days prior to his admission here, he had seemed improved, but on the third day prior to admission his fever rose to 105°F., he had a convulsion, and for a brief time no spontaneous breathing occurred. During the mid-afternoon of that day, a second episode of respiratory arrest took place. An endotracheal tube was inserted, and oxygen therapy was started. The infant was given a 2.5 per cent glucose in Ringer's lactate solution by clysis. Antibiotic therapy consisting of Combiotic and streptomycin was instituted. On the second day prior to admission, he became comatose. His breathing was irregular, and the diarrhea ceased. A cut-down of the medial malleolar vein of the right ankle was made for the administration of a 5 per cent glucose solution. On the day prior to admission here, he had a body temperature of 92°F.

At the time of admission at University Hospitals, the patient was unresponsive except to painful stimuli. His pupils responded sluggishly to light. Inadequate spontaneous breathing was present. The endotracheal tube was in place, and he was receiving artificial respiration. The heart sounds were faint, and the pulse was rapid. No blood pressure could be recorded. Evaluation of the chest was difficult, but there seemed to be a decrease of breath sounds in the left chest. The abdomen was distended. The skin was cool and mottled, and a moderate degree of edema was present.

Artificial respiration was given by means of a bag and mask for a time, and then a Bryd artificial respirator was employed. Two hundred fifty milliliters of a solution containing 5 per cent glucose and 15 mEq. of sodium chloride was started as intravenous therapy in the cut-down. One gram of chloramphenicol and 1,200,000 units of penicillin were added to the solution. Five hundred milligrams of sulfadiazine was given intravenously. Levophed was started, and in about 45 minutes the blood pressure had risen to 80/40 mm. Hg.

The urinalysis showed a pH of 6.0, albumin 4+, sugar 2+, and blood 1+, and microscopic examination revealed 0 to 4 white cells and 1 to 4 red cells per high power field. The hemogram revealed 13 Gm. hemoglobin and 74,150 white blood cells, and a differential count of 44 band polymorphonuclear leukocytes, 28 segmented polymorphonuclear leukocytes, 14 lymphocytes, 10 monocytes and 4 unclassified cells.

A lumbar puncture was done. The fluid obtained was slightly xanthochromic. Eight cells were present. The sugar value was 179 mg. per cent, and the protein value was 291 mg. per cent. A culture of the cerebrospinal fluid produced no growth.

The report on the serum electrolyte studies done at the time of admission revealed a CO_2 of 6.7 mEq./L., chloride 83 mEq./L., potassium 6.7 mEq./L., and sodium 108 mEq./L. Sodium bicarbonate was added to the intravenous fluid therapy.

Three hours after admission, the infant's vital signs seemed to stabilize, and radiographic examinations were obtained. An intrathoracic tube was placed in the second interspace in the left midclavicular line, and was put in under water-seal drainage. Repeat serum electrolyte values drawn at that time were: CO_2 17.5 mEq./L., chloride 70 mEq./L., potassium 4.9 mEq./L., and sodium 112 mEq./L.

The patient died 6 hours and 45 minutes after admission.

SUMMARY OF CLINICAL DISCUSSION

Dr. John C. MacQueen, Pediatrics: Mr. Thornwall will discuss the case for the junior students.

Mr. Carleton D. Thornwall, junior ward clerk: Today's protocol presents us with the problem of an infant who apparently had been well until he reached the age of eight months, when he developed gastrointestinal symptoms characterized by vomiting, associated with a flu-like disease.

On two subsequent occasions, diarrhea and fever developed, both bouts of diarrhea being severe enough to warrant his hospitalization. While hospitalized for his second bout of diarrhea, he showed initial signs of improvement, but then suddenly became critically ill, presenting a picture of high fever, convulsions, recurrent respiratory arrest, stupor, hypothermia and shock. On admission to this hospital, he was moribund, and in spite of vigorous therapy he died less than seven hours after admission.

In order to arrive at an adequate explanation for the course of the patient's illness, we had to postulate the existence of two separate entities. We felt that we must first explain the episodes of vomiting and diarrhea prior to the third day of his last hospitalization, and then attempt to explain his sudden deterioration and death.

Considering the initial course of events, we are faced with a differential diagnosis of recurrent vomiting and diarrhea in an eight-month-old infant whose past health had been good. Diarrhea at this age may occur from a multitude of causes which, for convenience, can be divided into about six groups.

Congenital mechanical defects such as gastrocolic fistula and malrotation can cause diarrhea, but we feel that these are very remote possibilities in this case, in view of the total lack of prior gastrointestinal difficulties.

Infectious diarrhea may be the result of bacterial or viral gastroenteritis, or may be secondary to parenteral infection. We also feel that this is unlikely, in view of the child's history of having twice improved markedly in response to what appears to have been no more than supportive and dietary therapy.

We also think that allergic diarrhea is a remote possibility, in the absence of any history of atopic symptoms in the patient and in his immediate family.

There is a possibility of an endocrinopathy, although it seems difficult to reconcile it with a history of normal growth and development. Therefore we tend to rule out hyperthyroidism or diabetes.

Cystic fibrosis of the pancreas is a possibility, and we are offered the tempting information that a two-year-old sibling has a chronic respiratory disease. However, we feel that this diagnosis is unattractive because there wasn't the usual picture of gastrointestinal dysfunction and chronic respiratory infection beginning shortly after birth.

Next, we come to the celiac disorders, and here we feel is the most likely explanation for the child's early illness. The onset of celiac disease usually occurs in the second six months of life, and is characteristically ushered in by an episode of vomiting and/or diarrhea accompanying an upper-respiratory infection. Following that initial flare-up, there usually are recurrent bouts of diarrhea. Each succeeding episode is more severe than its predecessor, until after the lapse of several months, the full-blown celiac picture emerges, with malnutrition, chronic diarrhea with pale, mushy stools, and general debilitation.

It is interesting to note that the first episode of diarrhea responded to dietary therapy, but the exact nature of that treatment isn't mentioned in the protocol. Children with celiac disorders usually respond dramatically to a gluten-free diet, and we assume that this was what happened in this instance.

We now are faced with the necessity of explaining the terminal series of events. We are told that after three days of apparent improvement, during his final hospitalization, the infant abruptly spiked a temperature of 105°F., convulsed and developed spontaneous respiratory arrest. He then became progressively less responsive, and later the same day developed a second episode of apnea. He was treated with tracheal intubation, oxygen, streptomycin, penicillin and parenteral fluids, with no apparent improvement in his condition. He developed marked hypothermia, and was transferred to this hospital.

On the patient's admission here, no blood pressure was obtainable. He was stuporous, spontaneous ventilation was inadequate, and breath sounds were diminished over the left chest. There was a marked leukocytosis with a shift to the left, 4+ albuminuria, glycosuria, and microscopic hematuria and pyuria. A spinal tap revealed xanthochromia, a normal cell count, and increased sugar and protein. Electrolyte study showed marked acidosis, with sodium and chloride depletion, and hyperkalemia. Chest films were taken, and on the basis of these a chest tube with water-seal was inserted in the left second intercostal space. We assume that this indicated the presence of either a tension pneumothorax or a pyopneumothorax. The patient was treated with Levophed, positive-pressure respiration, the aforementioned chest tube, large amounts of antibiotics, and electrolyte replacement. He improved temporarily, but soon expired.

We feel that the terminal picture was one of overwhelming sepsis, occurring in an infant already weakened by an acute diarrheal episode. The clinical findings suggest a pyogenic pneu-

monia, probably staphylococcal in origin, since it apparently had been contracted in hospitals and since it didn't respond to penicillin and streptomycin. We think that there probably was a terminal septicemia, and multiple mycotic microabscesses as terminal complications. To our way of thinking, this provides a tenable basis for the spinal-fluid findings, since minor subarachnoid bleeding commonly occurs with septicemia. We choose to ascribe the elevated cerebrospinal fluid sugar to the parenteral infusion. The urinary findings were also compatible with septicemia or even with prolonged diarrhea.

We attribute the glycosuria to dextrose infusions. It seems to us that the electrolyte picture can be explained on the basis of fluid and electrolyte losses secondary to the diarrhea, or more probably as secondary to adrenal damage due to the septicemia. We choose to explain the episodes of apnea either on the basis of so-called anoxic petechial hemorrhage into the brain stem during convlusions in an already hypoxic child, or mycotic abscesses in the same area. The pyopneumothorax or tension pneumothorax is characteristic of staphylococcal pneumonia occurring in approximately 25 per cent of all cases in infants.

In summary, our impression is early celiac disease with superimposed staphylococcal pneumonia and terminal septicemia.

Dr. MacQueen: That was a well designed discussion.

As the principal discussor of the protocol this afternoon, we are pleased to have Dr. Lee Forrest Hill, of Des Moines. To many of you, who know Dr. Hill, he needs no introduction, but for those of you who don't know him, I shall briefly list some of his professional positions and honors. He is past-president of the American Academy of Pediatrics, vice-president presently of the Iowa Medical Society, and physician-in-chief at Blank Memorial Hospital for Children, in Des Moines. More importantly, as far as this group is concerned, I can tell you that Dr. Hill is a fine gentleman, a scholar and a medical teacher unexcelled. He is the accepted and revered dean of pediatrics in Iowa.

Today, Dr. Hill, with our hospitality goes the task of discussing the protocol.

Dr. Hill: I must express my appreciation for being invited to participate in the conference today. When I last did so at S.U.I., some years ago, I was handed what I thought was a sort of "lulu." The patient was a boy who had been wounded in the thigh by a shotgun blast. Someone had poured turpentine in the wound, and a week later he developed bulbar paralysis and died. Botulinus organisms aren't supposed to grow in tissues and produce toxin, but in that instance they did, and the Pathology Department recovered them on culture. But when the pathologists made their report, my discussion proved not to have been completely misdirected. A similar case had been reported in the literature, and I had been able to cite it. Thus I had at least mentioned the possibility that turned out to be the correct one.

The case today is of considerable interest. From the information supplied in the protocol, we may conclude, first, that the baby had been in a normal state of health during his first eight months of life, and second, that he then developed three episodes of illness during a single month, the final episode culminating in his death.

Whether the three episodes had a common etiologic agent or were unrelated as to etiology can only be guessed at on the basis of the data available. However, a good "rule o' thumb" to follow in differential diagnosis is that one should assume that a single disease process, rather than two or more of them, explain the abnormal findings. In keeping with that bit of medical wisdom, I am inclined to take issue with the student discussor and to approach the problem with the supposition that the illnesses had the same etiologic basis, and that a single bacterial agent was responsible for all three.

The protocol indicates that the major systems involved were the respiratory tract, the gastrointestinal tract, the central nervous system and the electrolyte-water balance mechanism. How shall we relate the sequence of events that led to the infant's death? We are told that a two-year-old sibling had had some type of respiratory disease since birth. Could he have been the source of his younger brother's illness? It would be helpful to know whether the other members of the family, including the mother and father, had had abscesses or boils or other evidence of a staphylococcic infection. Had the two-year-old ever been in a hospital where he might have picked up a resistant staphylococcic infection? Such family infections have been documented by a number of observers, especially in relation to hospital nursery staphylococci, where the infant brought the infection home to his family, and the chain of infection so started then continued to smoulder and flare up in various ways for a year or more.

When I read the opening paragraph of the protocol, my first thought was that the two-year-old had cystic fibrosis of the pancreas, and that that was probably the explanation of the problem in the nine-month-old, too. We're not told whether a sweat test had been done on the two-year-old, but the description of the patient's good progress during the first eight months of his life doesn't support such a suspicion. For the same reason, celiac disease, even in an early stage as suggested by Mr. Thornwall, seems unlikely. The bulky, foul-smelling stools, distended abdomen and wasted muscles characteristic of the malabsorption syndrome were all absent in this patient.

I doubt that we need to attach much significance to the brief period of cyanosis that followed the breech delivery. The fact that the baby was well

enough to go home on the third day and progressed normally from then on excludes brain damage as playing any role in the subsequent events. Likewise, I think we may conclude that the baby didn't have a congenital defect—for instance adrenal hyperplasia of the salt-losing type, as might have been suggested by the first set of electrolyte values obtained after the infant's admission to this hospital, or a renal defect of the salt-losing type. His progress seems to have been too uneventful and normal for these or other types of congenital abnormalities to have been present.

Now we come to the first of the three episodes in the baby's illness. He developed a fever with vomiting which was diagnosed as being due to "flu." Flu covers a multitude of sins. We all use it as a wastebasket diagnosis when we aren't sure of anything more specific, and perhaps that was the situation here. The baby could, of course, have had a viral infection. It would be helpful to know what the white blood count and differential were, and what a throat culture and an x-ray film of the chest showed. Also, one would like to know what antibiotics were used—if any. This could have been an infection with a staphylococcic organism, for staphylococcic pneumonias are notorious for recurring, and one always has the task of deciding when to stop treatment in such cases.

We are told the baby was well only for a few days. Then he became ill again with fever, bronchitis and, this time, diarrhea. Was the same etiologic agent responsible for this and for the first and subsequent episodes? It seems reasonable to suppose so, and one suspects that it was bacterial, rather than a repetition of a viral infection or "flu." The diarrhea could have been due to a specific bacterial enteritis, and one's suspicions would be directed largely toward staphylococci, pathogenic E. coli or Salmonella organisms. Or it might have been parenteral—i.e., a diarrhea secondary to an infection elsewhere in the body, and in this case most likely somewhere in the respiratory tract. Again we have no stool cultures, blood counts or chest x-ray films to give us a clue as to the nature of this episode, but the fact that the diarrhea subsided in a few days following only dietary therapy suggests that bacterial enteritis wasn't present at that time. Presumably, the respiratory infection cleared too, for we are told that the baby appeared to be in a normal state of health for the next 10 days. Mr. Thornwall listed a number of possible causes for the diarrhea with which I agree, but he suggested the possibility that removal of gluten in the dietary management might have been responsible for the subsidence of the diarrhea. Although I suppose it could have happened, I should consider it a rather remote possibility.

Then the baby developed his third and final episode. This time he was really sick. He had a recurrence of his fever, and the diarrhea was much more severe. He passed 20 to 30 stools per day. At this point we need to know something of the character of those stools. Were they copious and watery, or merely dabs of stain on the diaper? If copious, a severe degree of dehydration would appear to have been inevitable unless counterbalanced by fluids administered by some other route. We haven't been given anything other than the baby's birth weight, but we might assume that a normal nine-month-old infant would weigh in the neighborhood of 9 Kg. A deficit of 60 ml. of water per kilogram, or approximately a pound of body weight, would probably produce no symptoms. If the deficit had been twice that much, or 120 ml./Kg., however, the baby would have lost two pounds, or 10 per cent of his body weight, in which case he would surely have shown symptoms from dehydration. And if his water deficit amounted to 160 ml./Kg., or a weight loss of three to four pounds, he would undoubtedly have had severe symptoms from dehydration, probably with circulatory collapse, shock and possibly death. Hence, knowledge of the baby's weight at that stage of his illness, in comparison with his weight before the diarrhea, would be most helpful in evaluating his degree of dehydration.

Also, it would be helpful if we knew the nature of the oral feedings that were continued during the diarrhea. Dehydration may be hypertonic, isotonic or hypotonic. In hypertonic dehydration, more water than electrolyte is lost. Serum sodium values are above 150 mEq./L. For years it has been customary for most of us to order B.S.M. (boiled skimmed milk) in the dietary treatment of diarrhea. But now it is known that milk, Lytren or other electrolyte-containing fluids given in excess may contribute materially to hypernatremia or hypertonic dehydration. In isotonic dehydration, water and electrolyte losses are of similar degrees, but in hypotonic dehydration, more electrolyte than water is lost. Serum sodium values are less than 130 mEq./L. The giving of large amounts of electrolyte-free fluids such a water contributes to this type of dehydration.

At this stage in the baby's illness, we can only conjecture as to the type of dehydration he might have had—if, indeed, he was dehydrated at all. But if we glance ahead at the serum electrolyte values obtained when he was admitted here, it is clear that at that time he was in severe hypotonic dehydration with metabolic acidosis.

Three days before admission, the baby's temperature rose to 105°F., and he had a convulsion and stopped breathing. What caused that convulsion? Again, we have to speculate. Could it have been due to the high fever—a febrile convulsion? Febrile convulsions usually occur shortly after the onset of the hyperpyrexia, and are of short duration. Moreover, they are not attended by the serious manifestation of interference with breathing that occurred in this case. The possibility of a febrile convulsion could, I should think, be dismissed. Could the baby have had a menin-

gitis as part of a general sepsis? No mention is made of the respiratory tract in the description of the third illness that has been furnished us. Were there signs of pneumonia? Was the spleen enlarged, and was the baby jaundiced? Platou and his associates at Minnesota reported on 16 cases of sepsis in infants, and found the same organism in the spinal fluid and blood in six of the 16. I think I would have been suspicious at this point that the baby might well have a meningitis as part of his infectious process to account for the convulsion. The signs of meningitis in a nine-month-old infant may not be the same as those in an older child. The nuchal rigidity and positive Kernig's sign may be absent. Examination of the spinal fluid is most important. That wasn't done in this case until the baby arrived at this hospital. The cell count at that time was only 8, and the culture was sterile—findings which effectively exclude the possibility that the cause of the convulsion could have been meningitis.

There remain two other possible causes for convulsion that I should like to mention. One is thrombosis of cerebral blood vessels due to marked hemoconcentration and sluggish circulation. The reported hemoglobin value of 15 Gm. suggests hemoconcentration. The expected level for infants of this patient's age would be in the range of 10 to 11 Gm. The xanthochromia and high protein reported in the spinal fluid suggests the possibility of hemorrhage. However, neither hemorrhages in the optic fundi or paresis of cranial nerves or other abnormal neurological findings have been described, and thus one can only mention the possibility of such a complication. If we had reason to believe that the baby had hypertonic dehydration or hypernatremia, we might consider the type of intracranial bleeding in this condition described by Finberg.

But there is still another possibility that interests me. If the baby had hypotonic dehydration at the time of his convulsion—and he certainly did when his electrolytes were first determined at this hospital—then the occurrence of a convulsion strongly suggests the possibility of water intoxication, with a swollen brain resulting from the disparity between the electrolyte and water content of body fluids. To make such a diagnosis, however, it would be necessary for us to know what the electrolyte values were at the time of the convulsion. Had this information been secured, and if the same pattern had been found to be present two days later, then the proper fluid for intravenous administration would have been hypertonic saline in a 3 per cent solution. If one estimates how much sodium is necessary to elevate the serum sodium from 108 mEq./L. to perhaps 135 mEq./L., on the basis of body water and weight, he finds that the amount is something like 125 mEq. of sodium. Instead, the infant received subcutaneous fluid by clysis and, later on, 5 per cent glucose water parenterally, both of which probably added to his difficulties rather than relieved them.

Now we come to the admission of the child to this hospital. Clearly, the baby was in shock. Perhaps "moribund" would best describe his condition. Voluntary respirations were absent, presumably due to the inability of the respiratory center to respond. Blood pressure was zero. The pulse was faint and rapid. The skin was cold and mottled. All of these findings have been described in patients under hypotonic dehydration. The distended abdomen and the cessation of diarrhea could be explained by an ileus secondary to the infant's serious toxic state. Hypopotassemia is another possible cause. Although the serum potassium was elevated, it may not have been an accurate reflection of intracellular potassium content. An electrocardiographic tracing might have given a more reliable indication of the potassium status.

My curiosity is aroused by the comment that there seemed to be a decrease in breath sounds in the left chest. We aren't told what the percussion note was. If it was flat, a pleural effusion would have been a possible explanation for the decreased breath sounds, but if it was hyperresonant, a pneumothorax was probably present. The fact that a tube was placed in the chest under water-seal drainage after an x-ray had been obtained confirms a tension pneumothorax. Thus, the possibility of a staphylococcic pneumonia is again raised. Could we look at the x-ray at this time?

Dr. John Keller, Radiology: This x-ray shows a very extensive tension pneumothorax on the left side, with depression of the left leaf of the diaphragm. The right side shows a vague infiltrate in the mid-lung field.

Dr. Hill: You couldn't tell from this film whether or not there had previously been a pulmonary infection produced by a staphylococcus?

Dr. Keller: I should say that there is no x-ray evidence of previous pulmonary infection.

Dr. Hill: What had been the source of air in the pleural cavity? An endotracheal catheter had been put in, and it may have been the source of the pneumothorax. Is there nothing to show whether there was a rupture of an emphasematous bleb or a pneumatocele?

Dr. Keller: I can see no evidence of a bleb or pneumatocele.

Dr. Hill: Well the question as to why the child developed a tension pneumothorax remains unanswered. Was there pneumonitis, pneumatocele, emphysematous bleb, ruptured abscess, or a traumatic fistulous tract made so that air kept pumping into the pleural cavity? Perhaps that point won't be cleared up until we get to the postmortem findings.

The urinary findings are not too different, I think, from what one might have expected in a very ill baby. The high white blood cell count with a marked shift to the left certainly speaks

for an infectious process, as well as for hemoconcentration. The spinal fluid findings, with the high protein and xanthochromia, suggest intracranial bleeding. I shouldn't expect to find a protein of 291 mg. per cent in water intoxication, but the rest of the story fits so well with hypotonic dehydration and water intoxication that I dislike to abandon the idea. I'll be much interested in what was found at postmortem.

Consequently, I'd like to take just a bit different view from that which was taken by the first discussor. I would sum up my thinking by saying that the baby had an infectious process and that the same infection accounted for all three episodes of illness. This might have been a staphylococcic infection ending in a pneumothorax from a ruptured pneumatocele, an emphysematous bleb or possibly a small abscess. The diarrhea, I should presume, was secondary to the infection. Cultures of the stools during the diarrhea would have been helpful in determining whether or not a specific enteritis—perhaps staphylococcic—was present. Certainly the diarrhea resulted in dehydration—most likely hypotonic dehydration. With such a severe diarrhea, I don't think we need to consider other causes of hypotonic dehydration such as cystic fibrosis, salt-losing renal disease or adrenal hyperplasia of the salt-losing type. If the baby did have hypotonic dehydration, then I think the convulsion might well have been on the basis of water intoxication. I shall be interested, as I said, in whether the brain has been found to weigh a little more than normal or to be waterlogged, or whether there has been some hemorrhage to explain the xanthochromia and high protein.

Dr. MacQueen: Before further questions are asked, I shall tell you that the infant's parents gave very little information about the weight of the baby or the number of stools. There weren't any informants who could help us very much.

Dr. Ian Maclean Smith, Internal Medicine: I have been wondering about three points that might count against a staphylococcal infection as the total diagnosis here. The diarrhea apparently occurred out of the blue. There had been no preceding operation, preceding starvation or preceding coliform suppression by antibiotics. The second point is that I don't see any evidence of pseudocysts on chest x-ray, and I wonder whether this may not have been unusual for staphylococcal pneumonia in a child. Third, supposing staphylococci were the cause of the cerebral inflammation —and I don't remember seeing this happen in the absence of steroids or endocarditis—I wonder whether there is any mention of pyocyaneus on the patient's chart, or whether there is any evidence anywhere of the presence of blue-green pus.

Dr. MacQueen: The answer to all of those questions is "No."

We'll have the report by the pathologist. There are more surprises to come.

Dr. Daniel Longnecker, Pathology: There are a rather limited number of autopsy findings, but I think we have all that are necessary to explain the clinical course. An infectious process apparently was the oldest disease process present. It was a bilateral otitis media, with mastoiditis. At autopsy, both the middle ears and the mastoid air cells were described as containing a purulent exudate bilaterally. We don't know how old the process was, but we assumed that it had been present throughout the patient's illness and that it was the basic disease process. There was no evidence of extention of the infection from the middle ear. That is to say, there was no evidence of meningitis.

A more striking feature, but secondary to the otitis, was a severe cerebral edema with softening of the brain and microscopic evidence of neuronal damage. The brain was heavy, weighing about 200 Gm. more than would be expected in a child of this size. On microscopic examination of sections from the cerebral cortex, we found scattered neurons in which the cytoplasm was somewhat eosinophilic. That is the change on which we base our assessment of neuronal damage. The Purkinje cells in the cerebellum similarly demonstrated some cytoplasmic eosinophilia. There also was some alteration in the staining quality of their nuclei.

The cerebral edema and the neuronal damage probably were the result of cerebral anoxia that had occurred during a convulsive episode. The seizures may have been on a febrile basis, but the proposal that they were secondary to dehydration, with electrolyte imbalance, is probably more likely. Death was attributed to the severe brain damage.

There were a few incidental findings. The most notable of them was thrombosis of the venous system of the right kidney. This was evidently of very recent origin. There was no evidence of organization of this thrombus, and its age was estimated at about one day. The kidney was congested, but there was no evidence of infarction. The urinary findings noted clinically were probably on this basis.

The possibility of pneumonitis has been suggested repeatedly. Although there was one area of early pneumonitis, it was too limited in extent to have contributed significantly to the morbidity.

There was evidence of generalized edema and ascites. I have no specific explanation for these, but I suppose they may have been related to fluid therapy. We also saw evidence of the pneumothorax. As the chest was opened, the left lung was found to be collapsed, and the right lung was inflated.

Cultures were obtained at autopsy, and two different organisms were grown from the two ears. In the right, Escherichia coli was found, and in the left, Aerobacter aerogenes. Pseudomonas

aeruginosa was grown from the spleen. There was no growth from the blood. The fact that there were three different organisms grown from three different, single sites makes it difficult to assign significance to any one organism.

Dr. Frederic W. Stamler, Pathology: Have you found anything to account for the pneumothorax?

Dr. Longnecker: No, I have no concrete suggestion. The explanation that occurred to me was that a needle had been put into the heart for injection of intracardiac adrenalin. I think that the people who have had more to do with artificial respiration might suggest that the pneumothorax was a complication of that procedure.

Dr. Wallace W. McCrory, Pediatrics: Was there any mediastinal emphysema?

Dr. Longnecker: None was described.

Student: Do you have any explanation for the diarrhea?

Dr. Longnecker: The gastrointestinal tract showed no changes. I don't believe we did a culture. We assume that it was a complication of the otitis.

Dr. Robert T. Soper, Surgery: Were the adrenals normal?

Dr. Longnecker: Yes.

Dr. Hill: This postmortem diagnosis reminds me of the time, back in 1928 in the era of Dr. Marriott, of St. Louis, when all of the children with severe diarrhea who lost considerable weight overnight were said to have cholera infantum and were operated upon for mastoid whether the ear showed much or not. I think the idea spread to this vicinity, too, when Dr. Jeans came up from St. Louis. For quite a little time, the middle-ear cholera infantum diarrheal syndrome and mastoid operation had a considerable vogue. It has gone out of existence since, only to be revived by today's protocol in an infant who had abscessed ears and diarrhea, and died as those infants used to do. I don't know whether they all died after the operation or not, but I did want to comment that the cycle goes round and round. Here we are again where we were 40 years ago.

Dr. MacQueen: Dr. McCrory has written about hypo-osmolarity and related subjects. Perhaps he would like to discuss this case.

Dr. McCrory: When Dr. Hill began, he mentioned that the last previous time he came to discuss a CPC at this hospital, he had a "lulu." I'm not sure what we ought to call this one.

What Dr. Hill stressed was the vital importance of knowing the specific answers to many questions that we didn't have specific information about. Hence, we cannot effectively discuss the management of the early stages of this child's disease. As far as I am concerned, we can take our pick of whether he was "hypo" or "hyper." But we have no choice as regards accepting the fact that events occurring to this infant in association with diarrhea led to his death. That is why we spend many, many hours talking about parenteral-fluid therapy and about the importance of knowing the particular disturbance that exists in a particular patient at a particular time.

I think the other thing that impressed me as I listened is that medical problems today involve somewhat more than the disease that the patients present when they are admitted. Frequently there are iatrogenic complications. We have to concern ourselves not only with what the infection itself is going to do to the patient, but also with the alterations that will occur in the patient as a result of his therapy. I mention this because most observers have concluded that the pneumothorax on the left was a result of a preceding staphylococcal infection. It is my impression, after looking over the chart, that this pneumothorax was actually the result of the therapeutic efforts to keep the patient breathing, and that it wasn't really related to staphylococcal infection. Similarly, I think that the disorder that the child developed in the previous hospitalization was a complication of therapy that could be intelligently analyzed only if we had what we do not have—some estimate of blood electrolyte concentrations at that particular time.

I wish to stress to you that this is a most vivid example of the importance of finding the cause for infection resulting in diarrhea, and secondly, of knowing what the electrolyte disturbance is in a particular patient before any fluid therapy is carried out. The only opinion I would have is that oral therapy for a child having profuse diarrhea is in all probability ineffective. I must immediately qualify that statement by adding that it depends upon what kind it is, but oral therapy for an infant *already depleted and dehydrated* won't result in replacement of effective circulating blood volume, and it won't prevent the development of dehydration, thrombosis and central nervous system damage. It would appear that this was a factor in this case.

Dr. Charles H. Read, Pediatrics: Would you like to say anything about the use of clyses in dehydrated babies?

Dr. McCrory: Not anything good, Dr. Read. You will notice that the solution used was Ringer's lactate in 2.5 per cent glucose. That is an isotonic electrolyte solution, and it would be fine if the patient were diluted. It would only have compounded the difficulty if this infant were already hypertonic. This is one of the means that has been used in experiments with animals to produce central nervous system damage, convulsions and death by producing a hypertonic condition.

Now to answer the specific question of whether clyses can be used. There will be instances, probably, in the lives of all of us when we are unable to perform an intravenous. That circumstance is the only one in which I think it justifiable to turn to clysis until the patient can be brought to

the point at which a cut-down can be put in. I think that a solution that contains electrolyte in approximately half the concentration of saline, and 2.5 per cent glucose, if one must guess, is the best solution one could use. Be aware that a child who is on the verge of shock, or who is in shock, will not mobilize fluid given by clysis. Thus, it can be completely ineffective, and in fact it may pull electrolyte and fluid out of the patient into the area to make it isotonic with the rest of the body fluid. Consequently, clyses can contribute to the development of further circulatory imbalance.

Dr. Samuel J. Fomon, Pediatrics: What is believed to be the significance of the organisms cultured from the spleen at necropsy?

Dr. Longnecker: I refer the question to Dr. Stamler, who is more acquainted than I am with departmental philosophy.

Dr. Stamler: I'll give you my own personal philosophy on it. I think that in general we tend to rely too much on postmortem bacteriologic reports. The report that says pseudomonas organisms were grown from the spleen or some other organ doesn't necessarily mean very much. Many types of organisms may gain entrance to the blood stream in the agonal state of an individual's life, and these can be cultured from various organs. Unless we know the number that were cultured and correlate that information with other things, our information doesn't amount to much.

I'd like to comment also about the fact that certain organisms were cultured from the infected ears—Escherichia coli, I believe, from one of them and some other organism from the other. I don't think this proves at all that these were the original offenders in this infection. Probably they were secondary invaders. Often such organisms do grow out in culture. They overgrow the culture, and some other organisms such as staphylococci or streptococci which might be present aren't recorded simply because they have been overgrown in the cultures. If one takes the material from such an infection and does a Gram stain, he may find these organisms that failed to grow out in the culture. In answer to the question that was originally posed, I think that the growing of pseudomonas organisms from the spleen in this case doesn't prove anything.

Dr. MacQueen: In brief review of the case, we can say that the child had an upper-respiratory infection that resulted in an ear infection. The ears were examined by the resident and a staff physician on call at the time of the patient's admission to this hospital. These men were not impressed that there was evidence of significant ear infection. This is a good example of how difficult some of these cases are to evaluate.

We assume that the child had a "parenteral diarrhea." This is a diagnosis that we haven't used for a long time, for it is no longer a fashionable one. Because pathogenic Escherichia coli and various viruses have been cultured from stools of some patients with diarrhea, a non-specific etiologic diagnosis has come to suggest a less-than-complete examination. Nevertheless, it is common experience that many infants and small children with the usual symptom of upper respiratory tract disease have diarrhea, and so the term *parenteral diarrhea* seems to remain valid. The severity of this infant's diarrhea was greater than usual. A tension pneumothorax was presumably caused by the artificial respiration.

The case has been discussed, but two final comments seem in order. Although we've made great strides in the treatment of the diarrhea of infants and children, it remains a potentially severe symptom. When it persists and/or recurs, it becomes important to establish the etiology. Second, there are great advantages, if one is to care for such patients, in having available the laboratory facilities to determine serum electrolyte values.

SUMMARY OF NECROPSY FINDINGS

The primary disease in this infant appears to have been bilateral otitis media with complicating mastoiditis. Although further extension to the cranial cavity might have been anticipated, there was no evidence of meningitis. Severe cerebral edema was present, and is assumed to have resulted from hypoxia occurring several days before death, during the clinically reported episodes of apnea. Neurons of the cerebellum and cerebrum exhibit changes that suggest anoxic damage, and it appears that brain damage was the cause of death.

Cultures made at the time of autopsy demonstrated Aerobacter aerogenes in material from one middle ear, and Escherichia coli in that from the other.

The thrombosis of the venous system of the right kidney was judged to be of no more than 24 hours' duration. Although there was congestion of this kidney, there was no infarction. The left kidney was uninvolved, and it is doubtful that renal insufficiency was significant in the terminal course. Albuminuria, glycosuria and blood cells in the urine were apparently signs of the venous thrombosis.

Incidental findings which were not thought to have played an important role in the terminal course included a mild early bronchopneumonia, focal interstitial pancreatitis, ascites and generalized edema.

ANATOMICAL DIAGNOSES

Bilateral purulent otitis media and mastoiditis
Severe cerebral edema, with anoxic neuronal changes
Venous thrombosis, right kidney
Left pneumothorax and pulmonary collapse
Generalized edema.

Coming Meetings

In State

Oct. 7 — **Radiology (Iowa Radiological Society and S.U.I. Department of Radiology).** Medical Postgraduate Conference. University Hospitals, Iowa City

Oct. 12 — **Northeast Iowa Clinical Conference.** Masonic Temple, Waterloo

Oct. 13-14 — **Arthritis and Rheumatism (Iowa Chapter of Arthritis and Rheumatism Foundation, Iowa Orthopedic Society, S.U.I. Division of Physical Medicine and Department of Orthopedic Surgery).** Medical Postgraduate Conference. University Hospitals, Iowa City

Oct. 13-14 — **Recent Advances in Urology (Iowa Urological Society and the S.U.I. Department of Urology).** Medical Postgraduate Conference. University Hospitals, Iowa City

Oct. 26 — **Fourth Annual Medical Legal Seminar (Webster County Medical Society and Webster County Bar Association).** Warden Hotel, Fort Dodge

Oct. 28 — **Mercy Hospital Medical Day.** Mercy Hospital, Des Moines

Nov. 8-9 — **Institute on Intrapartum and Neonatal Complications (S.U.I. Departments of Obstetrics and Gynecology, Pediatrics and Nursing; State Department of Health, Division of Maternal and Child Health).** Medical Postgraduate Conference. University Hospitals, Iowa City

Nov. 17-18 — **Current Concepts in the Diagnosis and Treatment of Cardiovascular Diseases (Iowa Heart Association and the S.U.I. Department of Internal Medicine).** Medical Postgraduate Conference. University Hospitals, Iowa City

Out of State

Oct. 1-4 — **Annual Session of the Colorado State Medical Society, combined with the Congress on Occupational Health (AMA).** Brown Palace and Shirley Savoy Hotels, Denver

Oct. 1-6 — **National Recreation Congress.** Cobo Hall, Detroit

Oct. 1-7 — **College of American Pathologists.** Olympic Hotel, Seattle

Oct. 2-4 — **Association of Medical Illustrators.** Hotel Kahler, Rochester, Minn.

Oct. 2-4 — **Obstetrics for Specialists.** Center for Continuation Study, University of Minnesota, Minneapolis

Oct. 2-5 — **American Academy of Pediatrics.** Annual meeting. Palmer House, Chicago

Oct. 2-6 — **Thirteenth Postgraduate Assembly in Endocrinology and Metabolism (Co-sponsored by The Endocrine Society and the National Institutes of Health).** Bethesda, Maryland

Oct. 2-6 — **Latest Application of Surgical Research and New Surgical Technics. 47th Annual Clinical Congress of the American College of Surgeons.** Conrad Hilton, Chicago

Oct. 2-6 — **Basic Electrocardiography.** Cook County Graduate School of Medicine, Chicago

Oct. 2-6 — **Vaginal Approach to Pelvic Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 2-6 — **Diagnosis and Therapy of Malignant Neoplasms of the Female Genital Tract.** New York University Medical Center, New York City

Oct. 2-13 — **Clinical Uses of Radioisotopes.** Cook County Graduate School of Medicine, Chicago

Oct. 3 — **American Association of Poison Control Centers.** Palmer House, Chicago

Oct. 4 — **Lederle Symposium.** Broadview Hotel, Wichita

Oct. 6 — **Physical Therapy in General Practice (U. of Nebr. Coll. of Med.).** Douglas County Hospital, Omaha

Oct. 6-7 — **Western Industrial Medical Association, Western Occupational Health Conference.** Biltmore Hotel, Los Angeles

Oct. 6-7 — **American Medical Writers' Association.** New York City

Oct. 6-7 — **First National Congress on Medical Quackery (AMA and the Food and Drug Administration).** Washington, D. C.

Oct. 7 — **Seventh Annual Meeting of the American Rhinologic Society.** (Meeting preceded by a 3-day workshop and seminar on dome preserving technics in surgery of the nasal tip, at the Illinois Masonic Hospital.) Belmont Hotel, Chicago

Oct. 7-9 — **International Symposium on Bone: Clinical Application of Recent Advances.** University of California, San Francisco

Oct. 8-13 — **American Academy of Ophthalmology and Otolaryngology.** Palmer House, Chicago

Oct. 9-12 — **Gallbladder Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 9-12 — **American Association of Medical Record Librarians.** Benjamin Franklin Hotel, Philadelphia

Oct. 9-13 — **Hand Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 9-13 — **The Physiologic Basis of Internal Medicine (American College of Physicians).** Duke University Medical Center, Durham, North Carolina

Oct. 9-20 — **Obstetrics, General and Surgical.** Cook County Graduate School of Medicine, Chicago

Oct. 11-14 — **Western Institute on Epilepsy, Thirteenth Annual Conference.** Granada Hotel and Inn San Antonio

Oct. 11-14 — **Diagnostic Cardiac Auscultation.** New York University Medical Center, New York City

Oct. 11-14 — **Mississippi Valley Conference on Tuberculosis.** Hotel Sherman, Chicago

Oct. 12-14 — **Urology.** University of California, San Francisco

Oct. 12-14 — **Congress of Neurological Surgeons.** Summit Hotel, New York City

Oct. 12-14 — **Academy of Psychosomatic Medicine.** Emerson Hotel, Baltimore

Oct. 12-15 — **Surgery of Hernia.** Cook County Graduate School of Medicine, Chicago

Oct. 13-15 — **Fifth Annual Convention of the American Association of Medical Assistants.** Holiday Hotel, Reno

Oct. 14-20 — **Second International Congress of Neurosurgery.** Statler Hotel, Washington, D. C.

Oct. 15-20 — **Fourth International Congress of Allergology.** Hotel Commodore, New York City

Oct. 16-20 — **Thoracic Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 16-20 — **National Safety Council.** Conrad Hilton, Chicago

Oct. 16-27 — **Internal Medicine.** Cook County Graduate School of Medicine, Chicago

Oct. 16-27 — **Basic Principles in General Surgery.** Cook County Graduate School of Medicine, Chicago

Oct. 17 — **Modern Methods of Evaluating Thyroid Function. (The Northwest Missouri Chapter of the Missouri Academy of General Practice and the University of Kansas School of Medicine).** The Moila Temple, St. Joseph, Missouri

Oct. 18-20 — **Council on Arteriosclerosis of the American Heart Association.** Hotel Balmoral, Bal Harbour, Miami Beach

Oct. 19 — **Annual Symposium on School Health (University of Kansas School of Medicine and the University of Kansas School of Education).** Battenfeld Auditorium, University of Kansas Medical Center, Kansas City, Kansas

Oct. 19-20 — **All That's New in Medicine. First Annual Milwaukee Medical Conference.** Milwaukee County General Hospital

Oct. 19-21 — **Dermatology for Specialists.** Center for Continuation Study, University of Minnesota, Minneapolis

Oct. 20-21 — **Central Neuropsychiatric Association.** Conrad Hilton Hotel, Chicago

Oct. 20-21 — **Dermal Pathology (University of Southern California).** Ambassador Hotel, Los Angeles

Oct. 20-24 — **Annual Meeting and Scientific Sessions, American Heart Association.** Americana Hotel, Bal Harbour, Miami Beach

Oct. 21 — **Symposium on the Problem of Obesity in Teenagers.** Stanford University

Oct. 22-25 — **American College of Gastroenterology.** Hotel Cleveland, Cleveland

Oct. 22-27 — **American Society of Anesthesiologists, Inc.** Statler Hilton, Los Angeles

Oct. 23-24	**American Cancer Society Annual Meeting.** Hotel Biltmore, New York City
Oct. 23-24	**Blue Shield Annual Program Conference.** Drake Hotel, Chicago
Oct. 23-27	**Advances in Surgery.** Cook County Graduate School of Medicine, Chicago
Oct. 23-27	**Gynecology, Office and Operative.** Cook County Graduate School of Medicine, Chicago
Oct. 23-27	**Clinical Cardiopulmonary Physiology (American College of Chest Physicians).** Sheraton Towers, Chicago
Oct. 23-27	**Introduction to Research Methods.** Armed Forces Institute of Pathology, Washington, D. C.
Oct. 23-Nov. 3	**Fractures and Traumatic Surgery.** Cook County Graduate School of Medicine, Chicago
Oct. 23-Nov. 3	**Urology.** Cook County Graduate School of Medicine, Chicago
Oct. 23-Nov. 4	**Laryngology and Bronchoesophagology.** University of Illinois College of Medicine, Chicago
Oct. 24-27	**American Dietetic Association.** Kiel Auditorium, St. Louis
Oct. 25-28	**American Association of Blood Banks.** Drake Hotel, Chicago
Oct. 25-29	**American Society of Clinical Hypnosis.** St. Louis
Oct. 26	**Lederle Symposium.** Hotel Pere Marquette, Peoria
Oct. 26-28	**Annual Course in Postgraduate Gastroenterology (American College of Gastroenterology).** Sheraton-Cleveland, Cleveland
Oct. 27-28	**Symposium on "Expanding Goals of Genetics in Psychiatry (1936-1961)."** New York State Psychiatric Institute, 722 West 168th Street, New York City
Oct. 27-29	**Association of Clinical Scientists.** Shoreham Hotel, Washington, D. C.
Oct. 28-29	**Problems Due to Infection in Medicine and Surgery.** University of California (Franklin Hospital), San Francisco
Oct. 30-Nov. 2	**Annual Assembly, Omaha Mid-West Clinical Society.** Civic Auditorium. Omaha
Oct. 30-Nov. 2	**Twenty-ninth Annual Assembly, Omaha Mid-West Clinical Society.** Civic Auditorium, Omaha
Oct. 30-Nov. 3	**General Surgery.** Cook County Graduate School of Medicine, Chicago
Oct. 31-Nov. 2	**Advances in Pediatric Diagnosis and Practice.** Medical College of Georgia, Augusta
Oct. 31-Nov. 2	**Interscience Conference on Antimicrobial Agents and Chemotherapy (American Society of Microbiology).** New York City
Nov. 1-2	**Fifteenth Annual Postgraduate Assembly.** San Diego County General Hospital, San Diego, California
Nov. 1-4	**American Society for Tropical Medicine and Hygiene.** Willard Hotel, Washington, D. C.
Nov. 2-4	**Diagnosis in Ophthalmology.** University of California, San Francisco
Nov. 3-4	**Central Society for Clinical Research.** Drake Hotel, Chicago
Nov. 4	**Problems of Adolescence.** Children's Hospital, University of California, San Francisco
Nov. 4-5	**Twelfth County Medical Societies Conference on Disaster Medical Care (Council on National Security and American Medical Association).** Palmer House, Chicago
Nov. 5-8	**Association of Military Surgeons of the United States.** Mayflower Hotel, Washington, D. C.
Nov. 6-8	**Gynecology (University of Kansas School of Medicine).** Battenfeld Auditorium, University of Kansas Medical Center, Kansas City, Kansas
Nov. 6-9	**EENT Problems (Section on Ophthalmology and Otolaryngology of the Southern Medical Association).** Dallas
Nov. 6-10	**The Internist's Role in the Pre and Postoperative Care of the Surgical Patient (American College of Physicians).** Mayo Clinic, Rochester, Minnesota
Nov. 6-10	**American Association of Inhalation Therapists.** Statler-Hilton, Buffalo, New York
Nov. 6-10	**Urologic Radiology for Radiologists.** Center for Continuation Study, University of Minnesota, Minneapolis
Nov. 6-11	**Electrocardiography.** New York University Medical Center, New York City
Nov. 6-17	**Surgical Technic.** Cook County Graduate School of Medicine, Chicago
Nov. 6-17	**Gynecology, Office and Operative.** Cook County Graduate School of Medicine, Chicago
Nov. 6-17	**Surgical Board Review, Part I.** Cook County Graduate School of Medicine, Chicago
Nov. 6-17	**Practical Cystoscopy.** Cook County Graduate School of Medicine, Chicago
Nov. 8-10	**Association of State and Territorial Health Officers.** Washington, D. C.
Nov. 9-10	**Theory and Practice of Contact Lenses.** New York University Medical Center, New York City
Nov. 9-10	**Review of Recent and Practical Problems in Medicine (University of Southern California).** Statler Hotel, Los Angeles
Nov. 9-11	**Symposium on Vascular Surgery.** New York University Medical Center, New York City
Nov. 9-11	**American Academy for Cerebral Palsy.** Chase and Park-Plaza Hotels, St. Louis
Nov. 10	**Fifth Annual Symposium on Diabetes (Chicago Diabetes Association).** Offield Auditorium, Passavant Hospital, 303 East Superior Street, Chicago
Nov. 11	**Postgraduate Conference on Allergy.** Presbyterian Medical Center, San Francisco
Nov. 11-13	**Alcohol and Civilization.** University of California, San Francisco
Nov. 13-16	**Interstate Postgraduate Medical Association of North America.** Cleveland Auditorium, Cleveland
Nov. 13-16	**Internal Medicine (University of Kansas School of Medicine).** Battenfeld Auditorium, University of Kansas Medical Center, Kansas City, Kansas
Nov. 13-17	**American Association of Public Health Physicians.** Cobo Hall, Detroit
Nov. 13-17	**Recent Advances in the Diagnosis and Treatment of Heart and Lung Diseases (American College of Chest Physicians).** Park Sheraton Hotel, New York City
Nov. 13-17	**Blood Vessel Surgery.** Cook County Graduate School of Medicine, Chicago
Nov. 13-17	**Surgery of the Cornea.** New York University Medical Center, New York City
Nov. 14-16	**Fractures in General Practice.** Medical College of Georgia, Augusta
Nov. 15-17	**Ophthalmology (Refraction) for General Physicians.** Center for Continuation Study, University of Minnesota, Minneapolis
Nov. 16-18	**Orthopedics for Orthopedic Surgeons and General Physicians (Hand Surgery).** Center for Continuation Study, University of Minnesota, Minneapolis
Nov. 16-18	**Milwaukee Divisional Meeting, American Psychiatric Association.** Hotel Schroeder, Milwaukee
Nov. 17-21	**National Society for Crippled Children and Adults.** Denver-Hilton Hotel, Denver
Nov. 18-19	**Psychiatry in General Practice (University of California, San Francisco).** Napa State Hospital
Nov. 21	**Hearing Problems (The Northwest Missouri Chapter of the Missouri Academy of General Practice and the University of Kansas School of Medicine).** The Moila Temple, St. Joseph, Missouri
Nov. 24	**Symposium on Anticoagulant Therapy.** University of Southern California, Los Angeles
Nov. 25-27	**American College of Chest Physicians.** Annual interim session. Brown Palace Hotel, Denver
Nov. 26-Dec. 1	**Radiological Society of North American, Inc.** Palmer House, Chicago
Nov. 26	**Third National Conference on the Medical Aspects of Sports (AMA).** Cosmopolitan Hotel, Denver
Nov. 27-29	**Annual Meeting of the American Society of Hematology.** Ambassador Hotel, Los Angeles
Nov. 27-30	**American Medical Association Clinical Meeting.** Denver Auditorium, Denver
Nov. 27-Dec. 1	**Surgery of Colon and Rectum.** Cook County Graduate School of Medicine, Chicago
Nov. 27-Dec. 1	**Advances in Medicine.** Cook County Graduate School of Medicine, Chicago
Nov. 27-Dec. 8	**Surgical Board Review, Part II.** Cook County Graduate School of Medicine, Chicago
Nov. 27-Dec. 8	**Obstetrics, General and Surgical.** Cook County Graduate School of Medicine, Chicago
Nov. 27-Dec. 8	**Practical Cystoscopy.** Cook County Graduate School of Medicine, Chicago
Nov. 30-Dec. 1	**Hematology.** University of California, San Francisco

MANAGEMENT OF THROMBOEMBOLISM

Two recent articles on the management of thromboembolism present the surgical interruption of the femoral vein as the treatment of choice. Since the introduction of anticoagulants some 20 years ago, many clinicians have considered them to be the preferable therapy for thrombophlebitis and thromboembolism.

In 1947, Edgar V. Allen and his associates at the Mayo Clinic reported their experience with some 2,000 patients.[1] From their study, those men concluded, "The expert use of anticoagulants, heparin and Dicumarol, has tremendously improved the outlook for patients who have vascular thrombosis." They continued, "Our experience with ligation of veins has been limited. That is the natural result of the gratifying experience with anticoagulants that we have had. It is to be remembered that the sole purpose of the ligation of veins is to prevent pulmonary embolism. Anticoagulants are used to prevent pulmonary emboli and to prevent extension of venous thrombosis. . . . Our carefully considered opinion after weighing all of the evidence, is that the use of anticoagulants is, in general, a much better method of treatment. We recognize a small role for ligation of veins, which is, at times, quite important."

In the second edition of their book on peripheral vascular disease, published in 1955,[2] the same doctors reiterated their position regarding the use of anticoagulants: "A large amount of statistical data supports the contention that anticoagulant therapy with heparin or one of the coumarin compounds, or both, is indicated in most cases of secondary thrombophlebitis, venous thrombosis or pulmonary embolism. The rationale is that anticoagulants limit extension of the thrombus and prevent thrombosis in other veins, and therefore prevent embolism. For postoperative patients with thrombophlebitis or pulmonary embolism, evidence indicates that recurrence or extension of thrombophlebitis and recurrence of embolism can be reduced almost to zero by adequately supervised and controlled anticoagulant therapy."

A report by Evans and Dee, of the Lahey Clinic, supported the recommendations of the Mayo men.[3] Results in 238 cases of postoperative venous thrombosis, they reported in 1948, "give us great confidence in trusting anticoagulant therapy in recognized cases of postoperative venous thrombosis."

Quite in contrast to these earlier reports, Byrne[4] concluded from a study of 979 cases of phlebitis at the Boston City Hospital that anticoagulant therapy is apparently of value in the prevention of pulmonary emboli, but is not so efficacious as carefully planned surgical therapy. The premise for preferring surgery is that anticoagulants do not dissolve thrombi but merely prevent their formation, and that one can accomplish little by administering them after clotting has begun. From his experience, he considers surgery the treatment of choice in the prevention of death from thromboembolism. In 979 cases of phlebitis there were 196 deaths—a surprisingly high mortality rate of 20 per cent.

The most recent report on the subject, based on a 20-year survey of experience at the Massachusetts General Hospital,[5] strongly recommends surgical management. Approximately two-thirds (2,122) of all the patients had been treated by femoral-vein ligation alone, and afterwards 12 patients (0.6 per cent) had fatal pulmonary emboli. Of 489 patients who had been subjected to femoral vein ligation and in addition had been given anticoagulants, one (0.2 per cent) died from embolism. In a group of 87 patients to whom anticoagulants had been administered some time after surgery, there were no deaths. Anticoagulants alone were given to 473 patients, and death from embolism occurred in 10 of them (2.8 per cent). Among the 103 patients who had anticoagulants first and surgery later, there was one fatal embolism (1.0 per cent). Vena cava ligation, with or without anticoagulants, was done on 22 patients, and all of them survived. From that experience, the authors conclude that the failure ratio of anticoagulant therapy was five times that of vein ligation.

The Boston doctors point out that much of the controversy over the problem of thromboembolism arises from the uncertainties of diagnosis. The thrombosis can be palpated or visualized no more frequently than the pulmonary infarction, and in many cases the diagnosis must be an inferential one. The differential diagnosis is often difficult, for local muscle strain, neuritis, arthritis, and cardiac and pulmonary abnormalities are hard to eliminate. "An awareness of the disease, careful history taking, alterations in the clinical chart, physical findings consistent with phlebothrombosis or pulmonary infarction, and occasionally roentgenography of the chest and electrocardiography," the authors declare, "remain the mainstays in diagnosis."

From their experience at Massachusetts General Hospital, they conclude, "At the present time thrombophlebitis of the femoral system, whether of spontaneous origin or after surgery, is considered to be most definitely, effectively and economically controlled by femoral-vein interruption. In most cases a bilateral procedure should

be done, but on occasion unilateral interruption affords adequate protection. If the phlebitic process has spread to involve the common femoral or iliac vein, thrombectomy and venous interruption are of little benefit, particularly in swelling of over two days' duration. Such veins are prone to rethrombose, and the hazard of subsequent embolism is great, particularly when simultaneous anticoagulants are not given." The most likely nidus for occult thrombi is the femoral venous system, and in the presence of pulmonary emboli, of which the source is not evident, bilateral common femoral-vein interruption is indicated. In those patients in whom pulmonary embolism occurs after femoral-vein interruption and the use of anticoagulants, vena cava ligation is recommended, though it may result in serious morbidity and a high mortality.

The residual chronic venous insufficiency that may be present after venous interruption is attributed to the ligation *per se,* by the proponents of anticoagulant therapy. The men who favor venous interruption insist, however, that the varicosities, edema, cellulitis and ulceration cannot be attributed to ligation, but are the result of preexisting thrombophlebitis or cardiac, renal or nutritional difficulties. The proper postoperative care of the patient and the care with which the legs are supported, they insist, play an important role in the prevention of disability.

The use of leg exercises by the bed patient, the bandaging of the lower extremities postoperatively, and early ambulation have reduced the incidence of postoperative thrombophlebitis greatly. Anticoagulants are at times employed prophylactically in patients who have undergone surgery and are especially likely to contract postoperative thrombophlebitis.

It is clear that two diametrically opposed methods of therapy for thrombophlebitis exist. The integrity of neither school can be questioned. The resolution of the problem can be achieved only through a fully controlled study in a large series of patients. Until the question is settled by that means, the individual physician must be guided by his training and by his own experience to employ the plan of therapy in which he has the greatest confidence.

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THERAPY FOR THE HAND

One-third of all injuries requiring the services of a physician or surgeon involve the hand. This is true in the office of the rural practitioner, as well as in the emergency room of the city hospital. What may appear to be a minor injury to the hand may involve a tendon or a nerve, and can result in a serious disability if not properly diagnosed and skillfully treated.

Paul R. Lipscomb* emphasizes that all physicians should be taught the basic principles that govern the treatment of an injury to the hand, so well defined by Kanavel, Koch and Bunnell. Wounds must be protected from contamination and infection, and in fact the emergency first-aid care may well be the most important phase of treatment. Before any attempt at treatment is considered, adequate facilities and help, and proper instruments should be at hand. Cleansing should be thorough and gentle. In contrast to wounds of the arm, leg or abdomen, debridement of the hand should be done sparingly. Almost all wounds of the hand should be closed primarily. Closure must be accomplished without tension, and skin grafts may be required to avoid tension. Fractures should be reduced and immobilized at the time of the primary treatment, unless the physician isn't trained to care for the injury adequately. Severed nerves and tendons do not require primary repair, particularly under unfavorable conditions. A physician who attempts primary suture of a flexor tendon in its digital sheath should have had considerable education, training and experience in reconstructive surgery of the hand, and should be capable of performing a tendon graft.

Lipscomb points out that extensive reparative, restorative, reconstructive and rehabilitative surgery of the hand should be carried out only in hospitals by well qualified surgeons who are dedicated to this field of surgery and who have been educated to work in orthopedic surgery, plastic surgery and neurosurgery. He must be thoroughly versed in the most minute details of surgical and functional anatomy of the upper extremity, in splinting and bracing, and in the rehabilitative procedures necessary to the achievement of good functional results.

Obviously, hand surgery is a very specialized field that requires strict attention to the basic principles of treatment, meticulous concern for detail, adequate training and experience, and dedicated service to the patient.

* Lipscomb, P. R.: Who should do surgery of hand? SURG., GYNEC. & OBST., **113**:233, (Aug.) 1961.

STRAPPING UMBILICAL HERNIAS IN INFANTS

In a report from Sweden, Karlström* strongly suggests that adhesive strapping of umbilical hernias in infants is unnecessary. In a 2½-year study of some 1,500 infants, he says he found 170 umbilical hernias, an incidence of 11.1 per cent. He classified 84 of them as small, 32 as possessing medium-sized orifices that admitted a finger tip, and 8 of them he regarded as large. Alternate patients were strapped with adhesive in a manner similar in every respect to that which is employed in this country. The other infants with umbilical hernias were untreated. Strapping was not started until the infant was between one and two months of age.

In the analysis of the results of treatment, 46 of the 170 infants were eliminated from the study group, either because healing had not taken place at the time of the report, or because of inadequate observation. Of the 124 cases analyzed, 56 had received adhesive plaster treatment and 68 had received no treatment of the hernia. In infants under four months of age, the incidence of healing was 46.4 per cent in the treated group and 45.6 per cent in the untreated babies. At six months of age, healing had occurred in 80.4 per cent of the treated cases and in 77.9 per cent of the untreated ones. After 12 months, 94.6 per cent of the treated group and 92.6 per cent of the untreated had healed.

The author quotes the report of Riisfeldt, from Copenhagen, who found that 24.7 per cent of the infants in his study had umbilical hernias during the first month of life. At three months and 12 months, the figures were 24.5 and 11.5 per cent, respectively. Among school children, Riisfeldt found the incidence of umbilical hernias at seven years of age to be 4.03 per cent, and at 14 years of age, hernias were present in only 0.25 per cent of the children.

Karlström concluded from his study that the strapping of umbilical hernias in infants is probably quite unnecessary, for most of them will heal spontaneously by the time the babies reach one year of age. The chief indications for the surgical treatment of umbilical hernias in children are (1) irreducible or incarcerated hernia, which is rare; (2) a large hernia; and (3) a hernia which is definitely increasing in size.

Gross** cautions that before initiating any measures for the treatment of umbilical hernia, it is well to remember that the majority of cases will be cured spontaneously as the children grow older and as the rectus muscles constrict and finally obliterate the hernial orifice. He recommends adhesive strapping during the early months of life, however, in order to augment the natural tendency for small hernias to vanish. If no appreciable benefit is observed by the end of six months, continuation of the strapping is useless. According to Gross, hernias 1.5 to 4.0 centimeters in diameter should be repaired, particularly if there has been recurrent discomfort, or a tendency of the hernia to increase in size. At the Boston Children's Hospital during the 25-year period from 1915 through 1939, more than 1,500 umbilical hernias were observed, and of that number, 360 were operated upon. From 1940 through 1951, a total of 439 umbilical hernias were treated surgically.

Despite the fact that the majority of umbilical hernias in infants will heal spontaneously with or without strapping, most mothers will prefer that the adhesive be applied, if for no other reason than that they dislike the appearance of a bulging navel. Physicians in this country have had confidence in this procedure, and will be loathe to discard it, despite Dr. Karlström's findings. On many occasions, relatively large hernias have become obliterated after several months of treatment.

* Karlström, F.: Should infantile umbilical hernias be treated with navel emplastra? J. Pediat., 59:87-89, (July) 1961.

** Gross, R. E.: The Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1953, pp. 423-427.

PREVENTION OF FARM ACCIDENTS

With the advent of fall, the Iowa farmer will be busily occupied with the harvesting of corn. As a result of the mechanization of farm operations, the toll of injuries and fatal accidents will be repeated, and the newspapers will again record the sad stories of carelessness with tractors, cornpickers and elevators.

Reports from N. J. Wardle, Ph.D., extension farm safety specialist at Iowa State University, reveal startling data about agricultural accidents in Iowa. In the 10-year period from 1947 to 1956, a total of 2,042 cornpicker accidents were recorded, with a peak of 412 mishaps in 1952. The number was reduced to 109 in 1956. During that decade, 1,391 persons lost 14 legs, 81 arms, 320 hands and 2,055 fingers in corn harvesting accidents. In the same ten years, 206 persons were injured in tractor accidents, and 402 farmers received injuries from elevators during the months of September, October and November. During those three months in 1958, there were 319 accidents, with 27 deaths, and during that quarter of 1959, a total of 275 accidents and 30 deaths were recorded.

Tractor accidents occur during all months of the year, but are commonest during the planting, cultivating and harvesting seasons. From 1947 to 1956, tractor-accident deaths took 454 Iowa farmers, and in the years 1957, 1958 and 1959, respectively, there were 50, 56 and 54 such deaths. In the first six months of 1960, there were 141 persons involved in tractor accidents, and 25 of them died.

The cornpicker is propelled by a tractor oper-

ated by a power take-off, or revolving shaft. The stalk of corn enters the V-shaped guide, the ears and husks are removed by revolving rollers, the stalk is ejected, and the ears of corn are carried to a hopper or wagon by means of a chain-operated elevator. A clogging or piling up may interfere with the efficient operation of the machine at any of several points, and it is when the operator attempts to unclog one of the devices that a maiming accident occurs. Entanglement in the power take-off results in many serious injuries. Tractor accidents result from the overturning of the machine, and produce crushing injuries.

Numerous factors help to cause accidents. Clearing the debris manually without shutting off the machine was regarded as the cause in 59 per cent of the cases. Hurrying was the next most common contributing factor (31 per cent). Fatigue, lack of a safety device, inadequate training, and the use of ill-fitting gloves or loose clothing have explained other accidents.

This year a bumper crop of corn is promised. Modern technics have allowed closer planting than ever before. Improved strains possess heavier stalks and produce higher yields. All of these factors add to the magnitude of the problem.

Inasmuch as the treatment of injuries of these sorts must devolve upon the doctors of the state, it is reasonable that the prevention of them also lies within the province of the medical profession. An accident-prevention program should be undertaken, and every means of communication should be utilized in promoting it. County medical societies might well initiate such campaigns, and their members should participate actively in them.

PSORIASIS

Psoriasis, according to Farber and Peterson,* occurs in from three to four per cent of the population. It may become evident at any age, but it is uncommon before the age of 10 years. It occurs with equal frequency in males and females.

These authors postulate the existence of a latent phase of psoriasis, a so-far unidentified condition that is present before the manifestation of the clinical disease. This latent phase may be present at birth or may develop later, but it is their contention that some underlying biochemical aberration is present long before the initial appearance of the skin condition. The stimuli that trigger the appearance of psoriasis initially, they think are the same as those that aggravate the clinical disease—the onset of winter, a gain in weight, acute streptococcic infection, dermal injury and emotional stress. The environmental, hormonal and genetic influences are unquestionably powerful, but their precise roles have never been made clear. Once clinical psoriasis develops, it is capable of widely varying severities but generally continues to be clinically evident. Complete remission to a latent state is uncommon, but it does occur. Exacerbations occur spontaneously or result from any of the numerous stimuli that have been enumerated.

In the therapy of 50 cases of psoriasis at the Stanford University Dermatology Service, the authors have found that the Goekerman regime of tar and ultraviolet light brings about a gradual resolution of the lesions. In one-fourth of the cases treated for periods of three to five weeks, there is gradual recurrence. In some three-fourths of the patients, the Goekerman regime resolves the lesions and a stable period follows, during which no additional lesions or only a few of them appear. This stability has lasted from five to 18 months.

The use of aminopterin, as reported by Edmundson and Guy,* does not alter the natural history of psoriasis, for upon the cessation of therapy there is a prompt return to the same clinical status. Farber also cautions against the use of atabrine and chloroquine, for 75 per cent of the patients with psoriasis develop a severe, generalized exfoliative psoriatic erythroderma about two weeks after the initiation of therapy with the antimalarial drugs. The corticosteroids will reduce the extent and severity of psoriasis, but upon cessation of therapy, the eruption recurs in much the same severity as had been present before the start of treatment. In about one-half of the patients, the post-steroid psoriasis is far more extensive than the pre-steroid condition had been. The eruption is often exudative and diffuse, and is accompanied by severe itching.

There is no magic cure for psoriasis, but the natural history of the disease can be altered by strict compliance with the Goekerman regime. Other methods of treatment with folic acid antagonists, antimalarial drugs or corticosteroids may have a deleterious effect upon the disease.

* Farber, E. M., and Peterson, J. B.: Variations in natural history of psoriasis. CALIFORNIA MED., **95**:6-11, (July) 1961.

* Edmundson, W. F., and Guy, W. B.: Treatment of psoriasis with folic acid antagonists. ARCH. DERMATOL., **78**:200-203, (Aug.) 1958.

NATIONAL FOUNDATION CAN'T PAY PHYSICIANS

William S. Clark, M.D., director of medical care for the National Foundation (formerly the National Foundation for Poliomyelitis) has asked the president of the Iowa Medical Society to help inform doctors of a change which his organization made in its policies and which seems not yet to be generally enough known. Following is the essence of Dr. Clark's letter:

". . . Prior to [1959], it had been permissible for National Foundation chapters to reimburse physicians for their services, and approximately one-third of our 3,100 chapters had been doing so in some degree.

"Our decision to eliminate such payments was influenced by several factors. We realized the overriding necessity of having uniform national policies, since it is quite common for physicians to treat polio patients from several chapter areas. We were also aware of the fact that physicians in some communities had been objecting to the practice of the payment of fees because of the problem of third party involvement and fee schedules which had not been worked out to everyone's satisfaction.

". . . We believe that the economic burden of catastrophic illness should preferably be alleviated by giving assistance for the burdensome ancillary costs of essential long-term or intensive care. Moreover, we believe that National Foundation assistance to families afflicted with such chronic diseases should be available to all who might suffer severe economic hardship from such an event. Thus our current policy permits assistance to families of private as well as non-private patients without involvement in the traditional patient-physician relationship, and without concern in the matter of the physician's and surgeon's fee.

". . . I should like to add that our staff would be happy to continue discussions of this complex problem and, if modifications of chapter patient-aid policies are desirable, such would be given serious consideration."

GRANTS FOR RESEARCH ON CHEST DISEASES

The Medical Research Committee of the Iowa Thoracic Society will accept applications until December 1, 1961, for research grants in the areas of tuberculosis, respiratory physiology and associated fields. Funds for research projects within Iowa are provided on a voluntary basis by county Tuberculosis Associations through the Iowa Tuberculosis and Health Association.

Members of the Medical Research Committee include Dr. Lewis J. Dimsdale, Sioux City, Dr. Jack M. Layton, Iowa City, and Drs. Ralph H. Heeren, Daniel F. Crowley and James F. Speers, Des Moines.

Application forms may be secured from the Thoracic Society Headquarters, 2124 Grand Avenue, Des Moines 12.

FLU VACCINATION URGED

Because of an expected rise in influenza this fall, vaccination against the infection without delay has been strongly urged by James W. Raleigh, M.D., medical director of the American Thoracic Society, medical arm of the National Tuberculosis Association.

Noting that Surgeon General Luther L. Terry of the U. S. Public Health Service had recently warned that there may be an upswing in the influenza cycle this fall, Dr. Raleigh pointed out that people who have tuberculosis, emphysema, and other respiratory ills in particular should take immediate advantage of the availability of an effective vaccine against influenza.

"When influenza strikes people who are already partially disabled by respiratory disease, the consequences may be extremely grave," said Dr. Raleigh. "It is urgent that they be vaccinated promptly against influenza, preferably before the fall months when the prevalence of influenza is expected to rise."

Dr. Raleigh added that the National Tuberculosis Association was alerting its affiliated associations and asking them to cooperate in every way possible with health officers and private physicians in the drive to vaccinate high-risk groups. These include, according to PHS, in addition to people with lung disease, those with heart disease, diabetes and other chronic illnesses, people over 65 years of age, and pregnant women.

OPEN MEETING OF AMERICAN PSYCHIATRIC ASSOCIATION

A divisional meeting of the American Psychiatric Association, open to all physicians and to personnel in related fields, will be held at the Hotel Schroeder, in Milwaukee, November 16-18. The registration fee will be $5.

Except for an opening address by Karl Menninger, M.D., the program will consist entirely of panel discussions. The topics are as follows: "Psychiatry and the Mental Health Associations," "The Isolated Psychiatrist," "Self-Appraisal of the Psychiatrist," "Problems of Treating Adolescent Patients in an Institutional Setting," "The Future of the Mental Hospital," "Correctional Psychiatry," "Legal Relationships in Psychiatry," "Psychiatry in the Schools," "Ethics in Psychiatry," "The Present Status of Drugs in Psychiatry," and "Symposium on Impulses."

Room accommodations should be arranged directly with Hotel Schroeder.

AN URGENT REQUEST

There are but few, if any, physicians who do not firmly believe in the free enterprise system. Though we find much to criticize in many areas of our American free enterprise, we see greater fault and danger in extensive governmental control of the pursuits of living.

Up until now, health care has been essentially a free enterprise in the United States. It is a $25 billion enterprise, $6 billion of which has been governmental, and $19 billion nongovernmental. Because the country's gross national product approximates $500 billion, transfer to government of the health care segment of our economy would not alone mean nationalization of our institutions. But a $19 billion item is significant. It is significant enough to be worth preserving.

Today we are all too close to a governmental hospital care program for the aged, to be financed through the federal social security system. There are, it is superfluous to say, political aspects to this question of government-sponsored health care for the aged; further, many very intelligent nonpolitical people support the social security proposal. It is said that passage of applicable legislation is no more than 18 months in the offing and might be considerably closer.

Two features of the proposed new social health care benefit are of dire import to the medical profession. For the first time there would be a broad-based popular tax levied specifically and solely for the purpose of paying for health care. In other words, we would have a limited compulsory governmental health program. The second, even more significant feature of the proposed program is that eligibility to receive the tax-paid care is not to be based on individual inability to pay, the traditional dividing line for all the population other than government wards. If a person belongs to a certain general category of the population, he will be eligible for governmental coverage whether he individually can pay for his own care or not.

The reasoning is this: of people over 65, those who can pay their own way are so exceptional that it would be impractical to make distinctions. But there are other marginal categories of people—the unemployed, widows, those who have had financial misfortune, those who have less than moderate income. As soon as the over-65 people are covered as a group for health care by government, there will be pressure for the government to protect at least some of these groups under age 65. Perhaps some should have tax-paid benefits. But the question will be where to draw the line.

Before long, we may be wondering if we can draw any line—any place. We are possibly about to have our last chance to perfect nongovernmental health care. We are in this critical situation because Americans, as have people in all other countries, have come to look upon adequate health care as an essential public service, a basic right. Here we come very close to demanding equal quality (albeit, perhaps, mediocre quality) for all people without regard for ability to pay. In 59 other countries, including all Europe except Finland, general compulsory governmental medical care is in operation to assure just this. And in America, we are moving on to satisfying this public service attitude toward health care. One way or another, it seems, we will get there.

Mr. Kennedy recommends that we, too, get there by shifting health care financing to government. Others suggest that we develop adequately our nongovernmental methods. Physicians as a group belong to the latter school of thought and, fortunately, most physicians realize that non-profit, community-oriented Blue Cross-Blue Shield is the best and perhaps the only way to solve the quantitative problems without jeopardizing qualitative performance. But the non-profit plans must be strengthened—and the medical profession must assume its indispensable role of leadership in this task. To use the words of Dr. Henry S. Blake, of Topeka, "Blue Shield—in this year, 1961—is the great unfinished business of American Medicine."

This, then, is a plea to the many exponents of the plans—the true friends of free enterprise—that they pick up the ball; that they more clearly delineate for their somewhat myopic colleagues the eleventh-hour, last-ditch aspects of the situation facing the medical profession; that they more convincingly establish that Blue Cross-Blue Shield is the best, if not the only, ally of the profession in its defense of free enterprise private practice.

This is an urgent request for prompt and purposeful action—by individual physicians and by their professional organizations.

—Richard J. Ackart, M.D., director of professional services of the American Hospital Association, in VIRGINIA MEDICAL MONTHLY, **88**: 489-490, (Aug.) 1961.

POSTGRADUATE COURSE IN GASTROENTEROLOGY

The American College of Gastroenterology will conduct its annual postgraduate course at the Sheraton-Cleveland Hotel, in Cleveland, on October 26-28. One complete session will be held at the Cleveland Clinic, and one at the Cleveland Academy of Medicine, and the faculty will be drawn from the medical schools of the area.

The subject matter will consist of the recent advances in diagnosis and treatment of gastrointestinal diseases. There will be comprehensive discussions of pancreatic disease, biliary tract disease, electrolytes, peptic ulcer, etc. In addition there will be an "X-ray Classroom" and instruction in cinegastrophotography.

For further information, address the College at 33 West Sixtieth Street, New York City 23.

IN MEMORIAM

Thomas Bentley Throckmorton, M.D., 1885-1961

Most obituaries are as cold as the CENTURY DICTIONARY definition: "An account of persons deceased; notice of the death of a person, often accompanied with a brief biographical sketch." The classic obituary of Thomas Bentley Throckmorton would read something like this:

Dr. Throckmorton died two months ago of a heart attack. He was born in Lucas County, in 1885, of a long line of country doctors. He attended Keokuk Medical College in 1904, then transferred to the Jefferson Medical College and was graduated with honors. One of the honors was the Dercum Gold Medal in Nervous and Mental Diseases. After a year at The Cherokee State Hospital, he moved to Des Moines, where he practiced the remainder of his life.

He and his talented wife reared four sons. One is a Des Moines surgeon, one a Des Moines lawyer, one is living at home, and one was killed in World War II.

During his lifetime, Dr. Throckmorton had many outside interests: He was a founder of the Des Moines Medical Library Club. He was Iowa governor of the American College of Physicians from 1920-1928. He was secretary of the Iowa State Medical Society for 14 years. He was secretary of the Interstate Post Graduate Medical Assembly of North America for 16 years. He was president of the Polk County Medical Society in 1952. He was chairman of the Neurological Section of the American Medical Association. He was grand master of Iowa Masons in 1936 and received the honorary thirty-third degree in 1945.

Dr. Thomas Bentley Throckmorton

Such facts, however, are merely the husks of the grain. The kernel or true worth of Tom's life lies buried beneath the statistics. In his 50 years of practice, he never refused a patient who could not pay. Many were down and out, and were befriended in addition to being treated for their physical ills. Tom lived in a "Small House" and raised his family in modest surroundings at a time when it was the style to own the "Big House." He taught his children the old-fashioned Christian virtues, and their accomplishments have proved the worth of Tom's lack of strut. He gave untold hours to organized medicine, without thought of personal aggrandizement, in an effort to raise the ethical standards of the profession he loved. He found, in his Masonic work, a soil for his love of ritual, companionship, and the fruition of his concept of a "way of life." He was profoundly serious and dedicated, behind his quiet twinkle, but he loved to tell "folksy" stories of the old country days, and he loved to go fishing. Perhaps his most treasured possession was a fishing trophy he received for catching the largest fish on a certain fishing expedition to the north woods.

It seems to me that a cold obituary is not enough. The obituary should also pick out the kernel of the life and hold it up to view. Tom's life was dedicated to fundamental Christian values and toward betterment of the profession he dearly loved. How many of us, in this busy work-a-day life, will be able to surpass him?

—DANIEL A. GLOMSET, M.D.

CONFERENCE ON DISASTER MEDICAL CARE

Representatives from local medical groups throughout the country are urged to attend and participate in the Twelfth County Medical Societies Conference on Disaster Medical Care, at the Palmer House, Chicago, on November 4-5. Physicians who wish rooms at the Palmer House should mention the conference in writing to the hotel, for a block of rooms has been set aside there by the AMA.

The program has been planned so as to emphasize medical preparedness at the local level and the training of the allied health professions. The following are some samples of the topics to be discussed: "Relationship of County and State Societies in Disaster Planning," "Health Mobilization in Total Warfare," "Rural Medical Support for Target-City Disaster," "Progress Report—Division of Health Mobilization, U.S.P.H.S.," "Progress Report—Committee on Disaster Medical Care, AMA," and "Expanded Training Programs."

President's Page

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President

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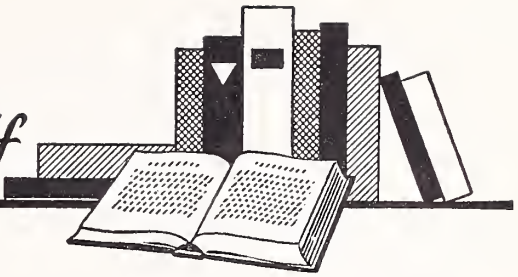


BOOKS RECEIVED

PATHOLOGY, FOURTH EDITION, ed. by *W. A. D. Anderson,* M.D. (St. Louis, The C. V. Mosby Co., 1961. $18.00).

ESSENTIALS OF NEUROSURGERY, by *Sean Mullan,* M.D. (New York, Springer Publishing Co., Inc., 1961. $6.75).

NURSING HOME ADMINISTRATION, ed. by *John D. Gerletti,* Ed.D., *C. C. Crawford,* Ph.D., and *Donovan J. Perkins,* M.S. (Downey, California, The Attending Staff Association, 1961. $6.50).

PROBLEMS OF PULMONARY CIRCULATION (CIBA FOUNDATION STUDY GROUP NO. 8), ed. by *A. V. S. de Reuck,* M.Sc., and *Maeve O'Connor,* B.A. (Boston, Little, Brown and Company, 1961. $2.50).

TRAITOR WITHIN: OUR SUICIDE PROBLEM, by *Edward Robb Ellis* and *George N. Allen.* (Garden City, New York, Doubleday & Company, 1961. $3.95).

THE CERVIX UTERI AND ITS DISEASES, by *C. Frederic Fluhmann,* M.D. (Philadelphia, W. B. Saunders Company, 1961. $14.00).

RESPIRATION IN HEALTH AND DISEASE, by *R. M. Cherniack,* M.D., and *L. Cherniack,* M.D. (Philadelphia, W. B. Saunders Company, 1961. $10.50).

BOOK REVIEWS

QUINONES IN ELECTRON TRANSPORT (CIBA FOUNDATION SYMPOSIUM SERIES), ed. by *G. E. W. Wolstenholme,* O.B.E., M.B., B.Ch., and *Cecilia M. O'Connor,* M.S., (Boston, Little, Brown and Company, 1961. $11.00).

At the very outset, it would appear worthwhile to state that this Ciba Foundation Symposium volume was prepared under the aegis of the Ciba Foundation, of Portland Place, Switzerland, the purpose of which is "to promote international cooperation in medical and chemical research."

The ubiquinone, or coenzyme Q, series of quinones are being investigated by more and more research workers. In this volume, the interdisciplinary views of 29 investigators have been pooled and collated. Although the work embraces the fields of biology, biochemistry and pharmacology, all of which are of interest to the medical practitioner, the presentations are sufficiently esoteric so that the book cannot be recommended to anyone other than a research worker in one of those scientific disciplines.

These quinones may someday prove useful as laboratory tools in attempts at determining the etiologies of certain diseases. For example, Dr. L. W. Wattenberg, of the University of Minnesota School of Medicine, has presented data on neoplastic disease which may indicate a trend. Thus, as often occurs in scientific investigation, results of the pure research described in this book may, in the long run, prove of inestimable practical value to the medical community.

This is not the type of book that can be read lightly or sporadically, but if one attacks it with interest and exerts some effort, the rewards will be great.—*Robert G. Martinek*

SYNOPSIS OF CONTEMPORARY PSYCHIATRY, SECOND EDITION, by *George A. Ulett,* M.D., and *D. Wells Goodrich,* M.D. (St. Louis, The C. V. Mosby Company, 1960. $6.50).

This small book of some 300 pages is exactly what it pretends to be—a synopsis of current psychiatric thought, diagnoses, clinical syndromes and therapies, and several tables on the recent developments and chemotherapy.

I strongly recommend this synopsis to the general practitioner who wants to gain a more scientific concept of the nature of current psychiatric practice, and I also recommend it to the members of ancillary professions such as social work and psychology.

I feel that this synopsis is particularly useful, also, to students preparing to enter the ancillary professions to which I just referred. It has been my experience that on many occasions such trainees are placed for practical training without having been given any very satisfactory understanding of the diagnostic and therapeutic methods with which they are immediately to be thrown in contact. Reading this book will give them a broad and general concept, but perhaps one that will be sufficient for their purposes.

I am enthusiastic about these practical possibilities. —*Howard V. Turner,* M.D.

THORACIC DISEASES, by *Eli H. Rubin,* M.D., and *Morris Rubin,* M.D., (Philadelphia, W. B. Saunders Company, 1961. $25.00).

This large volume, containing 968 pages and weighing 5 lbs. 12 oz., covers a large field. As its title indicates, though it deals mainly with the lung, it has a section on cardiopulmonary relationships.

Cardiac catheterization and angiography are discussed, but not cardiovascular surgery. For that matter, the book deals more with physiology and diagnosis than with surgical treatment. Though not ignored, surgical treatment is recommended where indicated, but not extensively covered. That is a good thing, for there isn't room for everything in one volume.

The 13 sections of the book have the following titles: I. "The Thorax and Its Contents," II. "Cardio-pulmonary Relationships, Circulatory Features," III. "Cardiopulmonary Relationships, Respiratory Features," IV. "Thoracic Disease in the Young," V. "Pneumonia," VI. "Bronchial Obstructive Disease," VII. "Intra-thoracic Neoplasms," VIII. "Tuberculosis," IX. "Occupational and Environmental Lung Diseases," X. "Thoracic Mani-

festations of Systemic Diseases," XI. "Diseases of Pleura and Subjacent Structures," XII. "Thoracic Injuries and Emergencies," and XIII. "Principles of Diagnosis."

I do not believe that the book is complete enough for reference work, but it is a good general volume. —*A. H. Kelly,* M.D.

THE CHANGING YEARS, by *Madeline Gray.* (New York, Doubleday & Company, Inc., 1958. $.95).

This paper-back volume has been written and revised by a very intelligent woman. The "modus operandi," unfortunately, involves a criticism of the medical profession that is none the less serious because it is used merely as a starting point. This woman's doctor failed to give her the solace and information she desired when she was going through the menopause. She had had a surgically-induced menopause, and had plied her surgeon with questions about her future. "But while he was a distinguished man and had done an excellent job," she says, "he was far too busy and to abrupt to bother with me. He answered that he'd done what he had to do, and that was that."

The result was that the author sought the desired information from lay individuals and medical authorities, and finally wrote this very pleasant book which includes such chapters as "Menstruation: Superstition and Fact" and "Don't Rush Into Hysterectomies." She writes in an entertaining fashion, she tells many stories, and she gives many examples and quotations. What she says makes good sense, and is easy reading.

The book is not one which a doctor needs for his library, but one which he might have in his reception room or might suggest as good reading for his distraught patients who are going through the difficult years of the menopause.

It is too bad that so many physicians don't have enough time to sit down with patients and reassure them regarding these disturbing questions, and that a lay person has to do the job for them.—*Daniel A. Glomset,* M.D.

MEDICAL-LEGAL SEMINAR

The Fourth Annual Medical-Legal Seminar, jointly sponsored by the Webster County Medical and Bar Associations, will be held at the Warden Hotel, in Fort Dodge, during the afternoon and evening of Thursday, October 26. Those who wish to attend must make advance reservations—no later than Saturday, October 21—by mail, sending their names (and their checks for $5 to cover the cost of the dinner) to Medical-Legal Seminar, 712 Snell Building, Fort Dodge.

At the afternoon session, doctors will question lawyers regarding "Malpractice in Iowa." A social hour and dinner will follow, and the evening session will provide lawyers an opportunity to question doctors about "Evaluation of Permanent Disabilities."

The presiding officers will be Dr. Otto N. Glesne, president of the Iowa Medical Society, and Mr. Lloyd Karr, vice-president of the Iowa State Bar Association.

MISSISSIPPI VALLEY CONFERENCE ON TUBERCULOSIS

The program of the Mississippi Valley Conference on Tuberculosis, October 11-14 at the Sherman Hotel in Chicago, will include a full day of medical sessions (Friday, October 13) sponsored by the Mississippi Valley Trudeau Society. The program consists of seven medical papers and two panels. Hour-for-hour Category I credit is to be granted to the members of AAGP who attend.

"The Use of Steroids in the Treatment of Tuberculosis" is the title of a paper to be presented by Dr. Robert H. Ebert, the ATS president, and Dr. John H. Hord, professor of medicine at Western Reserve University. The advantages of taking motion pictures of the fluorographic screen will be described by Dr. Joseph Jorgens, chief of the VA radiology service at Minneapolis. The results of a study of a new anti-tuberculosis drug, A-250, will be described by Dr. Arvine G. Popplewell, superintendent of Marion County General Hospital, Indianapolis.

Drug evaders, the headache of every doctor, are in for a surprise. Drs. John E. Kasik and Alfred Heller, of the University of Chicago, will describe a technic with which they are experimenting—a urine spot test—that will show whether the patient has taken his isoniazid. Staphylococcal pneumonia will be the subject of papers by Dr. Francis B. Landis, of the VA Center at Wood, Wisconsin, and Dr. Ian Maclean Smith, associate professor of internal medicine at S.U.I.

The analysis of respiratory gases through gas chromatography will be described by Drs. Lyle H. Hamilton, Ross C. Kory and Joseph R. Smith, of Wood, Wisconsin. Their experiments are directed toward measuring pulmonary capillary blood flow. Two panels will be concerned with "Childhood TB" and "Chemotherapy of Carcinoma of the Lung."

"The Increasing Problem of Drug Resistance" will be explored by Dr. James W. Raleigh, medical director of the National Tuberculosis Association, at a joint session of the Mississippi Valley Trudeau Society and Tuberculosis Association in the mid-afternoon of the same day.

COMMITTEE ACTIVITIES

The Iowa Medical Society's Statement to the Ways and Means Committee of the U. S. House of Representatives, July, 1961

(Dr. Wichern was given no opportunity to meet with the Committee, but his statement was accepted for publication as part of the testimony.)

Mr. Chairman and Members of the Committee:

I am Homer E. Wichern, a practicing surgeon; I reside at 300 Tonawanda Drive, Des Moines, Iowa. I am here in my official capacity as Chairman of the Legislative Committee of the Iowa Medical Society and as the official spokesman for the Board of Trustees of the Iowa Medical Society which has 2,424 members.

Throughout the century and more of Iowa's history, we doctors . . . together with the private, county and state hospitals . . . have met the health needs of economically-marginal elderly people, as well as the needs of everyone else who couldn't pay for his care. We have treated these people in their homes, in our offices or at local hospitals, either free of charge or at greatly reduced fees, and the local hospitals have provided them with beds and nursing care on a similar basis. It should be pointed out that in addition to this, the counties and the State of Iowa have long cooperated in providing ambulance service to and from Iowa City where hospitalization and specialist care at the University Hospitals was available not only for welfare clients with problem illnesses but also for ordinarily solvent people whom protracted health difficulties might otherwise impoverish. The University Hospitals is a centrally located health center which is publicly supported and each of the 99 counties has a liberal quota of available beds at this center.

As far as we know, no case has been called to our attention wherein these services were denied a resident of the State. Certainly this applies to the elderly group of individuals. It is our belief that the type of help which these people would find most acceptable would be a system under which they might budget, i.e. prepay, their emergency health care costs. For more than 20 years the hospitals have had available Blue Cross plans, and on April 12, 1945, we physicians started Blue Shield, which began writing contracts in September, 1945. These plans, together with those of several commercial insurance companies, furnish protection in somewhat varying degrees for three-quarters or more of the population of the State. Two years ago last spring, again in cooperation with the hospitals, the physicians prompted Blue Cross-Blue Shield to offer the "Senior 65" health insurance policy which carries a premium of $6.35 a month—$3.05 for Blue Cross and $3.30 for Blue Shield. Under this policy, physicians are entitled to no more than three-fifths of their customary charges and the hospitals provide a similar service at cost. A total of more than 12,000 policies of this type have been purchased to date, and though exact figures are not available, it seems certain that at least as many more have similar special policies from Mutual of Omaha, Continental Casualty and other commercial firms. The physician-sponsored Blue Shield plan has never, in its history, cancelled a policy because of age, and today more than 60,000 Iowans over age 65, including some over the age of 90, carry Blue Cross and Blue Shield coverage, which percentage is higher than when all other ages are combined.

Recent surveys in Iowa have revealed no prevalent unmet health needs among the elderly people. The most recent of them, the 15-county survey, was conducted last summer under the auspices of the Federal Department of Health, Education and Welfare. During interviews with the sampling of non-institutionalized elderly people in these 15 counties, no more than one in 20 of the respondents said he or she needed medical care or dental treatment but couldn't get it because of the cost. Iowa physicians, along with the other members of the American Medical Association, strongly supported passage of the Kerr-Mills Bill by the 87th Session of Congress. Last fall, at the urging of the Iowa Medical Society and a number of similar professional organizations, the 1961 General Assembly of Iowa passed a highly satisfactory Kerr-Mills enabling act (H.F. 470), designed to permit the establishment of whatever health-care plans may be found necessary for the near-needy aged in our State. This problem is still under study by the Legislature, and it appears that in due season the appropriation will be made when budget requirements of other necessary items are filled.

In Iowa, we are convinced, the Kerr-Mills Act has simply been delayed in taking effect, and it is our opinion that the Kerr-Mills approach to providing health care for the aged will come about in due season.

In the meantime, Iowa doctors are convinced it would be a serious mistake for Congress to enact the King-Anderson proposal (H.R. 4222) or any other plan for providing health services to OASI-benefit recipients. The following are our reasons:

First and foremost, such a scheme would indicate that for the first time, Congress would appropriate a *service* benefit in contradistinction to the cash benefits already in effect, the cost of which cannot be predicted. Secondly, such a scheme would do nothing for a considerable sector of the aged, which numbers approximately 100,000 people in Iowa. In this connection, I should like to quote from the report of the fifteen-county survey to which I made an earlier reference: "Among those past 75 [Social Security] coverage was considerably lower than in the younger groups (i.e., 65-70 and 70-75), and was especially low among the oldest women. Apparently many of the people past 75 had failed to qualify for Social Security coverage in time, and were left out of the program. We might note [and I am continuing to quote] that these people would not receive the benefits of the proposed program . . . unless coverage is extended to those who have been left out."

For these reasons, the physicians of Iowa respectfully ask you to reject the King-Anderson Bill (H.R. 4222, 87th Congress) and all similar proposals for attaching health care for all needy-aged to the Social Security System. We are sure that this Committee, in its wisdom, is aware of the fact that not enough time has been given to the implementation of the Kerr-Mills legislation (H.F. 470) and that the benefits of this Bill have not yet been brought to fruition. Instead, we ask that you allow sufficient time for the states to implement and test the adequacy of the Kerr-Mills Act.

INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION

The 1961 Scientific Assembly of the Postgraduate Medical Association will be held in Cleveland, November 13-16, and all licensed physicians will be welcome. The registration fee is $10, and luncheons and other special activities are extra.

The faculty will include the following doctors of medicine: Alton Ochsner, of New Orleans; F. Raymond Keating, Edward Rynearson and J. T. Priestley, of the Mayo Clinic; Edw. Maumenee and Allan C. Barnes, of Baltimore; Joseph Stokes, Jr., and Robert Dripps, of Philadelphia; Samuel F. Marshall, Neil W. Swinton and Wm. Dameshek, of Boston; Orvar Swenson and John L. Keeley, of Chicago; Charles Hufnagel, of Washington, D. C.; Wm. A. Oille, of Toronto; L. Reed Cramner, of Toledo; Harold L. Gainey, of Kansas City, Missouri; James M. Sutherland, of Cincinnati; and the following from Cleveland—Irvine H. Page, George Crile, Alan R. Moritz, Leonard L. Lovshin, Charles H. Herndon, Eugene F. Poutasse, F. A. Simeone, Benjamin Spock, Richard B. Stoughton, Herman Hellerstein, Roger B. Scott, Paul H. Curtiss, Jr., John Haserick, Louis Rakita, Irving Rothchild, Kingsbury G. Heiple, Herbert Johnson, Walter B. Pritchard, Edward J. Quilligan, George E. Spencer, Jr., Charles Rammelkamp, Oscar D. Ratnoff, Rupert Turnbull, Stanley Hoerr, Arthur Steinberg, Mason Sones, William Holden, Robin Anderson, James S. Krieger and Alfred Bochner.

Additional information and complete programs can be secured from the office of the Association, Box 1109, Madison 1, Wisconsin.

FILMS FOR COUNTY MEDICAL SOCIETY MEETINGS

The Iowa Tuberculosis and Health Association has announced the addition of the following four new professional films to its library. Professionally produced, they are both entertaining and well worth showing at county medical society meetings. All are 16 mm. sound films, and they are available on loan, without charge. Address the Association at 2124 Grand Avenue, Des Moines 12.

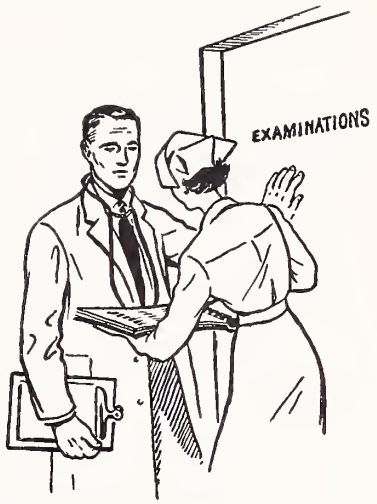
"Merry-Go-Round" shows a devoted doctor, pausing in the daily whirl of her duties to ask why tuberculosis is so tragically far from being defeated. Dramatically pushed back in recent years, this disease still attacks tens of thousands, still remains a hidden infection in millions, still causes suffering and death. How can people be made to listen? Who will do something about it? Dr. Jennings goes from politician to private doctor, from voluntary health worker to public health officer, from parents to ordinary citizens.

"World of Microbes" is a brilliantly photographed color film which demonstrates the phenomenon of phagocytosis. The battle between microorganisms and phagocytes is a continuous process that takes place in the body. When victorious, phagocytes are heroic in their efforts to control the infectious processes. If they lose the battle, phagocytosis may actually assist microorganisms in disseminating disease.

"Mississippi Valley Diseases." What causes histoplasmosis? How does it enter the body? Who are its victims? How widespread is it? These questions and others like them are answered authoritatively in this film.

"TB: Why Does It Strike?" When individuals are exposed to tuberculosis, some may become infected and contract clinical cases of the disease, and others may not. One factor which has a part in this selectivity is the nutritional status of the individual. In this film, Dr. Rene Dubos discusses his current research project at the Rockefeller Institute. The other participants are Charles Collingwood and "Red" Schoendienst, the professional baseball star.

THE DOCTOR'S BUSINESS



Make Provision for

A Corporate Trustee to Manage Your Estate

HOWARD D. BAKER
WATERLOO

The average doctor spends his productive years accumulating retirement capital and sufficient estate to provide for his family in case of his early death. Making those accumulations is a hard enough process, and taxes, bad investments and other occasional instances of poor judgment add to the difficulty. The decision-making, the planning and the worrying, in many cases, don't end with the doctor's death, but fall onto the shoulders of his widow.

Would your wife be able to assume financial responsibilities, and is it fair of you to expect her to do so even if she could acquit herself rather well? Many widows today would answer these questions with an emphatic "No!" This is the reason why more and more people today are arranging to have corporate trust departments manage their estates.

What do corporate trustees have to offer, how do they operate and what do their services cost?

1. A corporate trustee will manage all or any part of your estate, if you so direct in your will or in a trust agreement.

2. It will act as executor or trustee of your estate, assuring prompt action and proper know-how for best results.

3. Available funds will be fully invested at all times to produce maximum yields consistent with safety.

4. Changes in investments will be accomplished without worry or responsibility to your beneficiaries.

5. Your estate will be in one place for management purposes, thus doing away with your wife's having to deal with a number of individuals or agencies, and coordination will be provided at all times.

6. Your estate can be flexible enough to meet all contingencies, and still be kept within the goals that you have set for it, as regards investment policy and disbursement of funds.

7. Funds placed with a trust department are completely apart from other bank funds. National bank trust departments are subject to audit by federal bank examiners, adequate bonding is required, comprehensive records are required, and all investments must be approved by the trust investment committee, which is composed of senior bank officers.

8. Continuity of supervision is assured. This constitutes the chief advantage of the corporate over the individual trustee. An individual whom you might name may be fully competent, but death or disability might necessitate a less capable replacement.

9. Costs for executors are set by law. Fees for trust management average from one-half to one per cent of the annual value of the trust's assets. Thus, a $100,000 trust would cost about $500 per year to manage. Generally, that fee is repaid many times over through prudence in the management of the assets.

10. With taxes and inflation, the burden of conserving an estate is an enormous one. The size of your estate is not a significant factor. In fact, the smaller the estate, the bigger the job that has to be done if it is to accomplish the purposes for which it was established. Thus, competent supervision is especially important for small estates.

If you have reached the enviable position of having an estate that will be sufficient to achieve your goals, and if you have systematically planned it for minimum taxation, take another look at it from the standpoint of management. Have you provided for the most capable, continuous management—the type that will occasion the least worries and leave the fewest responsibilities to your loved ones?

Mr. Baker is a partner in Professional Management Midwest, and manager of its Retirement Planning Department. He majored in accounting and business administration at S.U.I., and was an agent of the U. S. Bureau of Internal Revenue for 3½ years before forming his present association in 1953.

AMA CLINICAL MEETING IN DENVER
November 26-30

The 15th annual clinical meeting of the American Medical Association will be held Nov. 26-30 at Denver, with a program geared to basic problems of medicine faced by physicians in their practice.

An outstanding scientific program, with emphasis on new research developments, has been planned under the direction of Samuel P. Newman, M.D., Denver, chairman of the AMA's Council on Scientific Assembly.

Some highlights will include sessions and papers on such important areas of medicine as genes and chromosomes, electronics and computers in medicine, space medicine, medical aspects of American habits, new developments in virology, treatment of radiation injuries, new findings in chemotherapy for cancer and latest data in the field of antibodies and antigens, Dr. Newman said.

With more and more nuclear reactors coming into use all over the nation, many practicing physicians soon may begin to face the problem of treating injuries from radiation accidents, the chairman said.

A section of internationally known experts in the treatment of radiation injuries will offer three major papers in this important new area of medical care. Chairman will be Marshall Brucer, M.D., chairman of the medical division, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tenn.

The radiation experts will discuss such topics as "Potential and Probable Sources of Radiation Accidents," "Diagnosis and Pathology of Radiation Injury" and "Treatment and Prognosis of Radiation Injury." Participants will include researchers from Los Alamos and Oak Ridge, the Office of the Surgeon General and the University of Chicago.

The age of advancing physical science also offers new findings to medical science: the use of electronics and computers in medicine. Chairman of this section at the Denver meeting will be A. H. Schwichtenberg, M.D., head of the department of aero-space medicine, Lovelace Foundation for Medical Education and Research, Albuquerque, N. M.

Computer systems for recording medical data to aid the physician in his diagnosis and prognosis will be discussed. Topics will include "The Future of Electronics in Medicine," "Microelectronics and New Concepts of Bioinstrumentation," "A System for Medical Data Recording," and "Biological Computers."

The virus, one of the most complex problems facing the clinician, will be the subject of a series of papers by outstanding specialists. Jonas E. Salk, M.D., Pittsburgh, originator of the killed virus polio vaccine, will give a paper on "Immunization Against Virus Diseases." Other topics will include "The Nature of the Virus and Its Cellular Reaction," "Smallpox Vaccination Complications," "Virus Hepatitis" and "Indentification of Viruses."

"We are confident that the 15th annual clinical meeting will offer one of the most interesting and informative programs ever presented at the winter session," Dr. Newman said.

"The program is designed to assist the physician in his practice. The latest findings in many areas of medicine will be presented by men who are top specialists in their fields. The meeting will be of great value to the clinician in advancing his knowledge."

POSTGRADUATE SHORT COURSES AT IOWA CITY

Three postgraduate courses will be held at the S.U.I. College of Medicine in October. The first, a one-day course in radiology sponsored by the Iowa Radiological Society and the S.U.I. Department of Radiology, will be held at the Medical Amphitheater on October 7. There is no registration fee for members of the Iowa Radiological Society, but the fee for non-members will be $10.00, which includes luncheon tickets.

On October 13 and 14, a postgraduate course, "Recent Advances in Urology" will be held in conjunction with a meeting of the Iowa Urological Society in the sixth floor operating rooms and E-405 at University Hospitals. There will be a $20.00 registration fee for non-members of the Society for this course.

Also on October 13 and 14, the Iowa Chapter of Arthritis and Rheumatism Foundation, the Iowa Orthopedic Society, the Division of Physical Medicine and the S.U.I. Department of Orthopedic Surgery will sponsor a course in Arthritis and Rheumatism, at the Medical Amphitheater.

AAGP credit will be given for all courses. Although accommodations are available in the University's Iowa Center for Continuation Study, for $3.50 per person per night, housing is always limited in Iowa City, and early registration is urged. The courses on urology and arthritis and rheumatism have been arranged to coincide with the Iowa-Indiana football game on the 14th, for those who are interested. Additional information and registration forms may be obtained by writing John A. Gius, M.D., Director, Postgraduate Medical Studies, College of Medicine, Iowa City, Iowa.

The following programs have been scheduled for the October postgraduate courses.

RADIOLOGY

Saturday, October 7, 1961

E. F. VAN EPPS, M.D., Moderator

8:30 Registration
9:00 Hydrometrocolpos
G. R. Barnes, M.D., Assistant Professor, Radiology
The Afferent Loop Syndrome
F. D. Lawson, M.D., Resident, Radiology

A New Cholecystographic Agent—Oragrafin
W. W. White, M.D., Resident, Radiology
Thermonuclear Warfare—Anticipated Fallout in Iowa
J. R. Thornbury, M.D., Resident, Radiology

9:45 Free Intraperitoneal Air in the Newborn
R. T. Soper, M.D., Assistant Professor, Surgery

10:15 Periureteritis Plastica
B. J. Begley, M.D., Assistant Professor, Urology

10:30 Appendicitis in Children—Radiographic Diagnosis
E. F. Van Epps, M.D., Professor and Head, Radiology

10:45 Au_{198} in the Treatment of Carcinoma of the Ovary
H. B. Latourette, M.D., Professor, Radiology

11:00 Progress Report of Lymphography
H. W. Fischer, M.D., Associate Professor, Radiology

11:10 Discussion

11:30 Lunch

12:30 The Diagnosis of Arachnoid Cysts
J. T. Keller, M.D., Assistant Professor, Radiology

12:40 The Lower Esophageal Ring
H. W. Fischer, M.D.
Treatment of an Intracardiac Tumor
D. C. Alftine, M.D., Assistant Professor, Radiology
Dermal Sinuses and Their Complications
B. J. Broghammer, M.D., Resident, Radiology

1:05 Bronchographic Differentiation of Neoplastic and Inflammatory Lesions
J. R. Thornbury, M.D.

1:30 Discussion

2:00 Film Session (Registrants are requested to bring interesting films for discussion.)

3:00 Business Meeting
Iowa Radiological Society

UROLOGY

Friday, October 13, 1961

8:30 Registration—6th Floor, University Hospital

9:00 Operative Clinic
R. H. Flocks, M.D., R. G. Bunge, M.D., D. A. Culp, M.D., B. J. Begley, M.D.

12:30 Luncheon—Doctors' Dining Room

Afternoon Session—E-405
R. H. FLOCKS, M.D., Presiding

1:30 Renal Function Following Ureteral Ligation
G. J. Bulkley, M.D., Associate Professor, Urology, Northwestern University Medical School, Chicago

2:30 Radiation Therapy for Carcinoma of the Bladder
R. H. Flocks, M.D., Professor and Head, Urology, SUI

3:30 Diagnosis and Treatment of Pyelonephritis
B. J. Begley, M.D., Assistant Professor, Urology, SUI

4:00 Some Variations of the So-Called Klinefelter Syndrome
R. G. Bunge, M.D., Professor, Urology, SUI

4:30 Problems Associated With Micturition
D. A. Culp, M.D., Professor, Urology, SUI

5:00 Business Meeting of the Iowa Urological Society

6:00 Cocktails and Dinner—University Athletic Club

Saturday, October 14, 1961

E-405, R. H. FLOCKS, M.D., Presiding

9:00 Pyelogram Clinic
Staff

12:00 Luncheon—Urological Seminar Room

1:30 Football: Iowa vs. Indiana

ARTHRITIS AND RHEUMATISM

Friday, October 13, 1961

8:30 Registration

9:00 Introductory Remarks

9:15 Gold Therapy in Rheumatoid Arthritis
T. B. Bayles, M.D., Research Director, Robert B. Brigham Hospital, Boston

10:00 Discoid L.E. and S.L.E., Differential Diagnosis
C. E. Radcliffe, M.D., Associate Professor, Dermatology and Syphilology, SUI

11:00 What the Inside of a Joint Looks Like Before and After Treatment
J. M. Layton, M.D., Professor, Pathology

11:30 Panel Discussion
T. B. Bayles, M.D., C. E. Radcliffe, M.D., and J. M. Layton, M.D.

12:30 Luncheon—Doctors' Dining Room

1:30 Temporal Arteritis
A. E. Braley, M.D., Professor and Head, Ophthalmology

2:10 The Treatment of Rheumatoid Arthritis in Children
R. D. Gauchat, M.D., Assistant Professor, Pediatrics

3:10 Roundtable Discussion on Treatment of Arthritis
W. D. Paul, M.D., Professor, Rehabilitation, Moderator

4:00 Question and Answer Session

6:30 Dinner: Curt Yocom's Restaurant—W. D. Paul, M.D., Presiding
Highlights of 1960 Big 10 Season

Saturday, October 14, 1961

Combined Program, Arthritis and Iowa Orthopedic Society

9:00 The Conservative Treatment of Arthritis
T. B. Bayles, M.D.

9:40 Surgical Rehabilitation of Arthritis
C. B. Larson, M.D., Professor and Head, Orthopedics, SUI

10:40 Progress Report on Finger Prostheses in Arthritis
A. E. Flatt, M.D., Associate Professor, Orthopedics, SUI

11:15 Demonstrations on the Home Care of Arthritis
W. D. Paul, M.D., T. B. Jones, R.P.T., and Kathryn J. Kelly, O.T.

1:30 Football—Iowa vs. Indiana

STATE DEPARTMENT OF HEALTH

Edward D. Zimmer
COMMISSIONER

CAT SCRATCH DISEASE

The first cases of cat scratch disease were recognized and described about 10 years ago. Since that time, cases of the disease have been reported with increasing frequency. There have been at least three in Iowa. At its July, 1961, meeting, the State Board of Health approved the addition of cat scratch disease to the Iowa list of reportable diseases.

The infection is called cat scratch disease because it usually follows a scratch from a cat. A few cases have developed after other incidents such as a prick from a porcupine quill, scratch from a splinter on a meat crate, handling wild rabbits, skin injury by beef bone, thorn pricks, mosquito bite, raccoon bite and a knife cut on the finger.

The causative agent of this disease is presumed to be a virus. There is no evidence that cats acquire the disease. Circumstances lead to the belief that the cat may act as a mechanical carrier of an agent acquired from some unknown source.

In the usual case of cat scratch disease, a firm red papule forms at the site of the scratch by a cat, and persists for some time. The regional lymph glands become enlarged about one or two weeks following the injury. Pus forms in one-third to one-half of the cases. Usually there is a low grade fever and some malaise. The enlarged glands frequently subside gradually and spontaneously in a few weeks. The illness is usually mild. In unusual forms of the disease there may be conjunctivitis, various exanthemata, encephalitis, myelitis, radiculitis and encephalomyelitis. It seems that the disease is most often observed in children, perhaps because they are more frequently scratched by cats. Ordinarily there is only one case per family, but Minnesota investigators* have reported multiple cases in some households. A review of 55 cases during a five-year period, from Toronto, Ontario,** suggests that the infection may be especially prevalent in some areas. Studies indicate that the highest incidence occurs during the fall and winter. Diagnosis may be difficult, and several laboratory tests may be required to rule out other diseases. There is no known specific drug treatment for this infection. Treatment often is unnecessary, for the disease is benign and self-limiting. In severe cases, aspiration of pus from the lymph nodes may shorten the course of the illness.

Currently, one of the problems is unavailability of skin test antigen, a useful diagnostic agent. This probably reduces the physician's ability to diagnose the milder cases. The State Hygienic Laboratory is attempting to make the antigen available to Iowa physicians.

* Warwick, W. J., and Good, R. A.: Cat scratch disease in Minnesota. AM. J. DIS. CHILD., **100**:228-247, (Aug.) 1960.

** Fowler, R. S., and Bailey, J. D.: CANAD. M.A.J., **84**:1365-1368, (June 17) 1961.

VIRUS OUTBREAK IN WEBSTER COUNTY

During the fall of 1960, because of the interest of the physicians of the Fort Dodge area in virus disease, the State Department of Health and the USPHS planned with Webster County health workers to set up a program for an around-the-year study of the different kinds of viruses that might appear in the community. The first large-scale virus outbreak in the Fort Dodge area since that time has recently made its appearance. A few cases occurred late in July, and a definite increase in incidence began during the second week in August. To date, it is thought that over 200 cases have occurred.

The illness is characterized by sudden onset, with headache, dizziness, fever of 103-104° F., and nausea. Many patients have abdominal pains, and others have muscle aches and pains. A larger share have herpangina, and a smaller number show a marked stomatitis. The duration of the illness is usually two to three days. A few individuals have recurrences. Although no patients have been dangerously ill, a small number have been affected severely enough to require hospitalization. Multiple cases are reported in a large percentage of families. The first case in a family is usually in a child. The incubation period seems to range from three to six days. Cultures obtained during the week of August 7 are showing virus growth.

Public health nurses and physicians are obtaining case histories and laboratory specimens from the affected individuals. To determine the extent of the illness, the percentage of persons in the area who are affected, the frequency of multiple family cases, the incubation period, the socioeconomic factors and other pertinent data, a random-sampling type of survey was set up for the week of

August 21. About 700 homes were visited to obtain the information. Eight investigators made the visits, calling at homes in about every eleventh block in the city of Fort Dodge. As this is written, the results are not yet tabulated.

The extent of the infection outside the immediate Fort Dodge and Webster County area will also be studied. It is already known that there are a number of patients in Humboldt County, to the north of Webster County.

Since the State of Iowa has no virus laboratories to which specimens may be sent, they are to be examined at the USPHS Regional Communicable Disease Center Laboratories, in Kansas City, Kansas.

This study has the approval and support of the Fort Dodge health officer and of the Webster County Medical Society.

ACCIDENT DEATH RATES DECREASE IN MOST STATES*

Every geographic division of the United States recorded a decrease in accident mortality in recent years. The greatest improvement occurred among residents of the East North Central area, where the accident death rate dropped from 61.8 per 100,000 population in 1949-1950 to 50.1 per 100,000 in 1957-1958, or about one-fifth, and nearly twice the relative reduction for the country as a whole. In the Pacific division, which had the second best record of improvement, the decrease was 14 per cent. Each of the eight states in those two areas recorded better than average reductions. In contrast, seven states—all of them in the South and Southwest—reported increases in accident death rates during that eight-year period.

Fatal falls have decreased in relative frequency throughout the United States. In every state but four (Louisiana, New Mexico, Nevada and Mississippi), the death rate from this cause was lower in 1957-1958 than in 1949-1950. Although the rate was reduced about one-fifth in the country as a whole, it decreased somewhat more than one-fourth in the North Central areas, and nearly as much in the West South Central and in the Mountain States. The death rate from falls continues to be highest in the northeastern section of the United States. In New England, accidents of this kind take more lives than any other type, including motor vehicle accidents. Inasmuch as the aged account for most of the fatalities from falls, the geographic distribution of the mortality from such injuries is influenced to a marked degree by the proportion of older people living in the various states.

The death rate from accidental drowning decreased 12 per cent in the country as a whole between 1949-1950 and 1957-1958, but this national average obscures wide geographic differences in trend. The greatest relative improvement occurred in the northeastern and midwestern sections of the country. At the same time, the frequency of accidental drownings increased in a belt of states extending southward from Tennessee and North Carolina and westward through Oklahoma. Currently, the highest mortality from this cause is recorded among residents of the Mountain and the South Atlantic States.

The trend of mortality from fire and from burns having other causes likewise showed marked geographic variations. In the period under review, the death rate from such accidents decreased one-third in the Mountain area, one-fourth in the West North Central and Pacific divisions, and one-sixth in the East North Central region. However, the trend has been upward in many southern states, notably Arkansas, West Virginia, South Carolina, Louisiana and Alabama, where the mortality from fire and from burns by other means has long tended to be relatively high. Most of New England also experienced an increase in the mortality from such accidents, yet the rate for the area as a whole is still below the national average.

In contrast to the other major causes of accidental death, the mortality rate from motor vehicle accidents increased in the majority of states between 1949-1950 and 1957-1958. Each of the 10 states in the Middle Atlantic and West North Central divisions, and six of the eight states in the South Central areas recorded a rise in motor vehicle accident fatalities. In Louisiana, the rate increased from 19.9 to 26.6 per 100,000 population, or one-third, and in Arkansas it increased almost as much. On the other hand, the death rate from motor vehicle accidents decreased in all the East North Central States, the reduction in Michigan being as much as one-fifth. Little change occurred in the motor vehicle accident death rate among residents of the Mountain States, where the rate is appreciably higher than that for any other geographic division. In New Mexico and Nevada, the mortality rate from such accidents was above 40 per 100,000 in 1957-1958.

* From the Metropolitan Life Insurance Company's STATISTICAL BULLETIN, June, 1961.

ELEVATED CHOLESTEROL IN HARDENING OF CEREBRAL ARTERIES

Elevated cholesterol levels have been found in persons who have suffered strokes as a result of hardening of the cerebral arteries, a group of researchers have reported in the September issue of ARCHIVES OF NEUROLOGY. Albert Heyman, M.D., and E. Harvey Estes, Jr., M.D., of Duke University, and M. Dean Nefzger, Ph.D., of the National Research Council, recommend, on the basis of their findings, that cholesterol-reduction methods be employed in managing patients with this condition.

The cholesterol levels of 68 men who had suffered this type of stroke were compared with the serum cholesterols of a comparable group of men

with no signs of hardening of the arteries. The mean cholesterol level in the stroke victims was "significantly higher," the investigators declared.

Although dietary restriction of fat and the administration of cholesterol-reducing agents haven't yet been made routine in the treatment of patients with hardening of the cerebral arteries, the belief that excessive blood-fat levels may be causative is not precisely new.

A STEP TOWARD A CURE FOR MULTIPLE SCLEROSIS

Research that may eventually lead to the suppression of such degenerative diseases of the central nervous system as multiple sclerosis and other similar neurological disorders was reported, early in September, at the Fourth International Congress of Neuropathology, in Munich.

Dr. Marian W. Kies and Dr. Robert Laatsch, of the National Institute for Mental Health, and Dr. E. C. Alvord and Cheng Mai Shaw, of the University of Seattle, reported having isolated the agent responsible for inducing experimental allergic encephalomyelitis (EAE), a disease in animals that resembles multiple sclerosis in man.

The causative agent, a small basic protein found in the nerve sheaths (myelin) of the brain, had previously been recognized as capable of inducing EAE in animals. Dr. Kies and her associates determined that the encephalitogenic activity of the highly purified water-soluble protein in the myelin is 100 times more potent in inducing the disease than is the brain tissue itself.

Significantly, this material was found to be capable of suppressing the disease, as well as of inducing it. The same protein that produces EAE when it is injected into the skin with an adjuvant, an emulsion containing killed tubercle baccili, was found capable of inhibiting the production of the disease when given without the adjuvant. In carefully timed and measured doses, either before or after the disease-producing inoculation, these preventive treatments have delayed or prevented the appearance of symptoms in animals otherwise doomed to die of paralysis, the investigators stated.

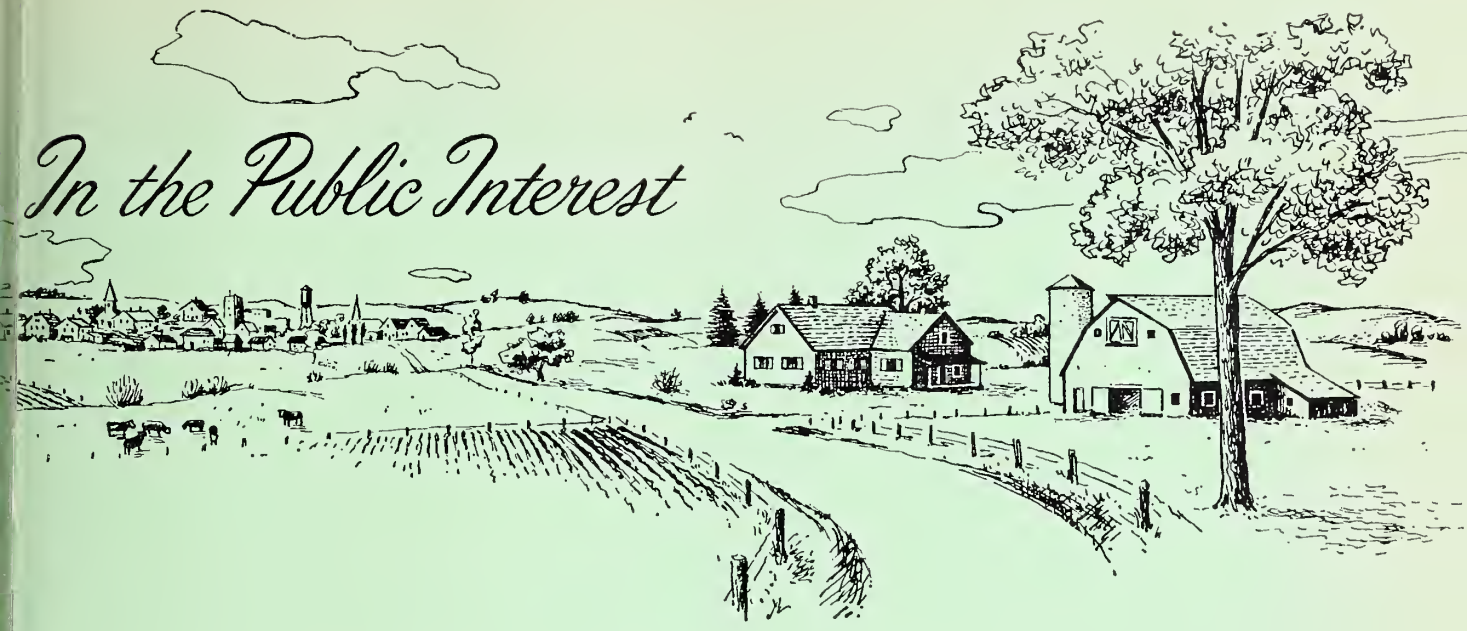
Until recently, it was thought that the only fractions capable of suppressing EAE were those that produced it, but recent studies suggest that treatment of the protein with formaldehyde modifies this antigen so that it no longer induces the disease but still retains its ability to protect animals against it.

In EAE, as in multiple sclerosis, lesions develop in the myelin of the central nervous system. The fact that the protein that damages the sheath of the nerve cell when injected with adjuvant is actually a constituent of the myelin sheath suggests that tissue auto-sensitization may account for the experimental disease.

The new findings in support of the theory that demyelinization may be induced by auto-sensitization provide important clues in the search for the initiating event that occurs in spontaneously demyelinating diseases. Further, the discovery that EAE can be suppressed by immunologic technics may provide an approach to the suppression of multiple sclerosis.

MORBIDITY REPORT FOR MONTH OF AUGUST, 1961

Disease	1961 Aug.	1961 July	1960 Aug.	Most Cases Reported From These Counties
Diphtheria	0	0	3	
Scarlet fever	44	104	59	Johnson, Polk
Typhoid fever	1	0	1	Pottawattamie
Smallpox	0	0	0	
Measles	63	236	29	Clay, Sac
Whooping cough	1	7	5	Pottawattamie
Brucellosis	10	29	13	Scott
Chickenpox	20	47	28	Scott, Story
Meningococcic meningitis	0	1	1	
Mumps	65	127	325	Boone, Des Moines, Scott
Poliomyelitis	6	1	4	Black Hawk, Des Moines, Marion, Polk, Wapello
Infectious hepatitis	95	159	28	Boone, Guthrie, Polk, Pottawattamie, Scott
Rabies in animals	32	53	21	Cerro Gordo, Franklin, Marshall, Poweshiek
Malaria	0	0	0	
Psittacosis	0	0	0	
Q fever	0	0	0	
Tuberculosis	25	35	35	For the state
Syphilis	83	74	92	For the state
Gonorrhea	109	111	133	For the state
Histoplasmosis	1	7	2	Polk
Food intoxication	0	0	0	
Meningitis (type unspecified)	12	1	1	Clay
Diphtheria carrier	0	0	0	
Aseptic meningitis	0	0	0	
Salmonellosis	5	2	5	Polk
Tetanus	1	1	0	Story
Chancroid	0	0	0	
Encephalitis (type unspecified)	0	1	0	
H. influenzal meningitis	0	0	0	
Amebiasis	3	2	2	Mahaska, Union, Woodbury
Shigellosis	2	3	2	Polk, Johnson
Influenza	0	0	0	



Doctors Must Take the Lead in

Perfecting Civil Defense Preparedness

The heightened sabre-rattling in East Berlin and in Moscow, and the new perils into which the United Nations has fallen, have led every husband and father at least to think about building a bomb shelter for his dependents. Physicians, though they are as concerned as everyone else for the protection of their families, have an additional and far broader responsibility to fulfill.

The AMA's REPORT ON NATIONAL EMERGENCY MEDICAL CARE and a subsequent pamphlet summary of it have stated the emergency obligations of doctors of medicine somewhat as follows:

1. To promote sound mass-casualty planning at all levels of government
2. To perfect their ability to manage mass casualties, regardless of their usual type of practice
3. To assure the full utilization of available medical and health resources—personnel, as well as supplies and equipment
4. To assure the prevention of unnecessary illness, injury and loss of life during and subsequent to a mass attack
5. To encourage the general public to engage in individual and collective survival training
6. To assure the maintenance of the health, physical stamina and morale of the uninjured survivors of a mass attack.

The coordination of civil defense planning in all of its aspects—the task referred to in the first of those six points—is the responsibility of government agencies, but federal and state officials must rely heavily upon the cooperation of the medical and the allied health professions. The statewide organization for the care of mass casualties in a disaster was worked out at least two years ago by the Iowa Interprofessional Association, at the behest of the Iowa Medical Society and in cooperation with the state civil defense director. A state committee on medical civil defense was formed at that time, a similar committee was set up in each of the eight civil defense areas into which Iowa is divided, and a county civil defense committee (composed, like the others, of six members representing the organizations that make up the IIA, the doctors, dentists, nurses, hospital administrators, pharmacists and veterinarians), was appointed in each of the state's 99 counties. Detailed plans according to which each physician and each nurse, nurse's aide, hospital administrator, dentist, pharmacist and veterinarian was assigned a responsibility, were worked out in some localities, and disaster drills were conducted in a few of them.

Now those plans must be reviewed and brought up to date, and drills should be held at intervals in all communities.

It should be pointed out that plans for treating large numbers of casualties can prove useful following natural rather than man-made catastrophes. They were activated only recently in the cities along the Texas coast. We don't have hurricanes in Iowa, but we have all too frequent tornadoes, and every once in a while a flood, a train or bus wreck, or a school, a picnic or some other public gathering struck by an explosion, a fire or an outbreak of food poisoning. Thus, even the least populous of our counties and the ones that

are farthest from a prime military target should prepare along with the rest.

DOCTORS SHOULD STUDY NEWLY DEVELOPED TECHNICS

The second of the AMA's points makes clear that each and every physician has an obligation to inform and otherwise prepare himself by every available means regarding the management of mass casualties. Inasmuch as most disaster victims have surgical problems and there won't be enough surgeons in every locality at the critical moment, the general practitioner and the nonsurgical specialist must be prepared to do any of the procedures that are indicated. The severely burned man, the woman with multiple compound fractures, the child with severe head injuries, and numerous panic-stricken people cannot be referred to the appropriate specialists in such a situation. The doctor who happens to be present and to be well enough to work—regardless of how he may previously have limited his practice—will have to do whatever is necessary.

In addition, there are some special sorts of administrative work for which all doctors need to prepare themselves. One of these is the assigning of priorities—"triage" or "sorting," as it is called. Shortages of personnel will necessitate "neglecting" some patients temporarily because their needs are less desperate than those of some others. Social prestige, possession of hospital insurance, or financial competence won't be considered. The vagabond with a severed artery will have precedence over the bank president with critical wounds of the gastrointestinal tract, or vice versa.

To inform himself about the principles and technics of "sorting" and caring for mass casualties, it is recommended that every physician study two publications intensively. The first, a booklet entitled SUMMARY REPORT ON NATIONAL EMERGENCY MEDICAL CARE, has already been mentioned. Member doctors can get it from the AMA on request. The second is the NATO handbook entitled EMERGENCY WAR SURGERY, and it can be obtained from the Government Printing Office, Washington 25, D. C., for $2.25 per copy. It presents some of the new concepts of sorting patients according to case priority, and reviews the principles of mass casualty medical treatment.

Procure these books, study them, and prepare yourself to help others survive!

GIVE LEADERSHIP TO DISASTER CARE TEAMS

Besides preparing themselves, physicians must take the lead in forming the members of the other health professions at the local level into teams that can start working together immediately and efficiently whenever disaster strikes. The representatives of the organizations that compose the Iowa Interprofessional Association were unanimous in deciding that physicians should head the medical civil defense committees at the state, area and county levels. Those doctors, in particular, should take action immediately.

Their first step should be to get their respective county medical societies to send them to the AMA's Twelfth County Medical Societies Conference on Disaster Medical Care, which is to be held at the Palmer House, Chicago, on November 4-5. A list of the topics that are to be authoritatively discussed there can be found on page 671 in this issue of the JOURNAL.

ADVOCATE SELF-HELP TRAINING FOR EVERYONE

Still another step is being planned—also one in which physicians are asked to take part. A program will be launched this month in which people will be taught how to meet their own health needs, if and when they are deprived of a physician's services. Called the "Medical Self-Help Training Program," it will be introduced to the medical and the other health professions by the U.S.P.H.S. in cooperation with the AMA. The subjects that are to be taught include: radioactive fallout and shelter; hygiene, sanitation and vermin control; water and food; shock; bleeding and bandaging; artificial respiration; fractures and splinting; transportation of the injured; burns; nursing care of the sick and injured; infant and child care; and emergency childbirth.

CONCLUSION

The AMA and the Iowa Medical Society are anxious to have all of their member physicians inform themselves thoroughly on the technics of mass-casualty surgery and medicine, and they urge all of the doctors who have been chosen to head local medical civil defense committees to take the lead in mobilizing both facilities and personnel for immediate service in the event of a natural disaster or a devastating military attack. Furthermore, they ask doctors to assist and advise in the setting up of self-help courses for the general public, and to conduct the classes themselves when they are requested to do so.

The time has come to get local teams ready for instant service. Apathy, as far as civil defense is concerned, must become a thing of the past.

Iowa Association of Medical Assistants

PROGRAMS, PAST AND FUTURE

At the beginning of last summer, each of our component societies was asked to report on those of its educational programs which had proved most interesting. Each of our program committees can get some valuable ideas from a review of the lectures and tours that have been selected as most worthwhile or enjoyable by the other organizations.

Susan Graham, president of the Oskaloosa District Association, the newest of our component societies, reported first upon a presentation by Dr. David O. Holman, an Ottumwa pathologist, on the subject of forensic medicine. He showed many slides, and asked his audience to decide whether the evidence contained in the pictures indicated that a crime had been committed. The second program that she discussed had been presented by the Mahaska County nurse, who showed a movie on mental health and told of the work being done in the Oskaloosa community in developing a program and a mental health clinic.

The Wapello County Association's report was written by Catherine Breed, of Ottumwa. Her organization's outstanding program had taken place in May, when the Fairfield Elks Club entertained the medical assistants of the area. The Fairfield Chamber of Commerce Chorus sang during the dinner, and the guest speaker was Fred Brandl, an exchange high school student from Austria. He showed colored slides of his homeland and explained the differences between the educational systems of his country and ours. His English is very good, and he speaks two other languages.

Mary Rhebb, of Sioux City, a past-president of the Woodbury County Association, said that her group's annual Civil Defense program had been a very interesting tour through SAGE installation at the Sioux City Air Base. At Woodbury's April meeting, Dr. Jerome Anderson, of the State Mental Hospital at Cherokee, spoke on the usefulness and needs of the River Heights Half Way House, a special residence for people with psychological and social problems. It was established to provide living arrangements for such people, intermediate between hospital and home.

Marjorie Snyder, of Anamosa, president of the Linn County Association, reported for Iowa's largest component society. The Cedar Rapids group has always had highly diversified programs, and choosing one or two of them on which to report was difficult. At one of them, four men representing different insurance companies engaged in a panel discussion on the ever-increasing amount of paper work that must be done in physicians' offices. At another, a panel from St. Luke's Hospital discussed hospital administration and the common problems of hospital administrators and medical assistants. The Wyeth Laboratories film "The Medical Assistant" was shown at the August meeting, and four physicians representing the Linn County Medical Society discussed it.

Dolores Mallinger, president of the Ft. Dodge District Association, reported upon two "terrific programs." In March, Dr. E. P. Eckart, a Ft. Dodge psychiatrist, had discussed "Human Behavior," preceding his remarks by describing his treatment room and its furnishings. (He has no couch!) The medical assistants were surprised to learn of the high percentage of teenagers in a psychiatric practice. Dr. Eckart showed a film, "Growing Toward Life," that indicated how the reactions and attitudes of others affect young people as they achieve adulthood. The second outstanding program had been presented in May by Mr. John Mitchell, a Ft. Dodge attorney. He spoke on "Malpractice," reminding his listeners of their responsibilities to their doctor-employers and to the patients as well.

Edris Clawson, past-president of both the Iowa Association and the Mason City organization, said that one of her group's most outstanding programs had been provided by her employer, Dr. John B. Dixon. He had performed eye surgery in India during the preceding winter, and he showed pictures of the people with whom he worked and the places where they live. The second program had been presented by Dr. Luke Chang, an internist is Mason City. Dr. Chang told about communism in China, emphasizing that it is not the choice of the people there, but is something that has been forced upon them. He also declared that a majority of the Chinese are still friendly to the West and loyal to Chiang, and that communism is doomed because it is in direct opposition to humanism.

Waneta Christensen, president of IAMA, forwarded the report of the Black Hawk Association. The Waterloo group's programs combined education with civic participation in a free immunization project sponsored by the Black Hawk Medical Society. On the last Saturday of each month, beginning last November, four nurses and four clerks assisted two physicians in administering polio vaccine, DPT immunizations and smallpox vaccine. More about this undertaking will be told in a future article devoted to civic activities.

Joyce Sweeting reported for the Iowa City District Association, of which she is the president.

Early this year, Mrs. E. W. Paulus told the medical assistants in Iowa City about the work of the Iowa City Senior Citizens Service. The second program, in August, had been a two-hour tour of the new municipal administration building in Iowa City. Lt. Beebee, of the Fire Department, had conducted the medical assistants on a tour of his facilities and then had given a talk on the role of the professional fire fighter in Civil Defense. Capt. Lee had guided the group through the various sections of the Police Department's facilities.

Margaret Hanson, president of the Scott County Association and president-elect of IAMA, wrote for Davenport. Her local group's most ambitious project had been the state convention, in May. In July, the members had been guests of the Scott County Medical Society for a panel discussion of the King-Anderson Bill, the discussants being a doctor, an insurance man and an attorney. At a later meeting, the medical assistants had had an opportunity to meet informally with state and Scott County welfare officials, the people with whom they deal every day regarding recipients of Old Age Assistance, ADC grants and county indigent assistance.

—HELEN G. HUGHES

KENNEDY'S FITNESS PROGRAM

According to Charles B. (Bud) Wilkinson, the University of Oklahoma football coach and special consultant on youth fitness to the President of the United States, the President's Youth Fitness Program consists of five recommendations:

1. That the preschool physical appraisal of children be broadened to include screening tests for strength, agility, and flexibility. These tests can be given in less than a minute, and in a very small space.

2. That pupils who fail the tests for strength, agility, and flexibility be required to participate in progressive developmental exercises and activities designed to build up muscular structures and enable underdeveloped students to meet minimal fitness levels.

3. That the screening tests be repeated every six weeks until all students reach minimum levels. Students with health limitations which preclude vigorous participation would be put on a program consistent with their physical condition. All medical and health needs should be attended to, of course.

4. That all students spend a minimum of 15 minutes per day participating in sustained conditioning exercises and developmental activities designed to build vigor, strength, flexibility, endurance, and balance. This, of course, is part of a broader physical education program.

5. That students be given a comprehensive test at the beginning and end of the semester. The first test would establish their present status and perhaps motivate them to improve their physical condition. Parents would have a clear understanding of where their child stands in the physical fitness scale. The second test would measure the child's progress made during the semester.

The AMA pointed out it is important that each school undertaking Wilkinson's fitness program make certain that it is carried out under the supervision of a physician. Careful preliminary physical examinations to make certain that there are no contraindications to physical training are urged for each student.

SUGGESTED FITNESS TESTS FOR CHILDREN

Strength can be tested by the pull-up, for boys, and the modified pull-up, for girls. Boys grasp the chinning bar with palms forward, and hang with arms and legs fully extended, and feet free of the floor. They then pull themselves up until their chins are over the bar. Then they lower themselves slowly until their arms are once again fully extended. Any boy should be able to do one pull-up. If he is 14, the minimum is two, and if 16, three.

The bar should be adjusted to chest level for girls. They extend their legs under the bar so that their arms hang, fully extended, straight down from the bar. The girl pulls herself up until her chest touches the bar, then lowers herself until her arms are again fully extended. Girls should be able to do a minimum of eight of these modified pull-ups.

Flexibility (and abdominal strength) can be tested by the sit-up for both boys and girls. The child lies on his back, with his legs extended and his feet about 12 inches apart. His hands are on the back of his neck, with the fingers interlaced. While someone else holds his ankles down, so that his heels remain on the floor, he sits up, twists his trunk and touches his left elbow to his right knee. Then he lies back down again, and the next time he sits up, he touches his right elbow to his left knee. Boys should do at least 14 of these and girls at least 10, in rapid succession.

Agility is tested by the "squat thrust" for boys and girls. It is started at the "attention" position, and is carried out in four movements. First, the youngster bends his knees and places his hands on the floor in front of his feet. His arms may be between, outside of or in front of his bent knees. Then in one movement he thrusts his legs backward so that his body is straight from shoulders to heels (the normal push-up position). Next, in one movement, he returns to the squat position. Finally, he returns to the erect position. This test should be timed. Boys should do a minimum of four correct squat thrusts in 10 seconds, girls three. One should make sure that the child returns to a fully erect position at the end of each squat thrust.

Children who pass these tests probably could pass a more elaborate physical achievement test.

Woman's Auxiliary News

OUR PRESIDENT SAYS—

Eligibility for membership in the Woman's Auxiliary to the AMA is unique in that only the wives of physicians belonging to the AMA and the widows of doctors who were members of the AMA in good standing at the time they died can qualify. There are over 2,400 women in Iowa who could meet one or the other of those requirements, but fewer than half of them are Auxiliary members. Perhaps you know a doctor's wife who hasn't given much thought to the importance of her taking part in the work of the Woman's Auxiliary. You can easily do a favor to her and to the medical profession by urging her to join.

Never in the course of history has there been a time when physicians' families have needed so much to work together in harmony at getting lay groups in this country and throughout the rest of the world to safeguard the ideals of the medical profession!

The Auxiliary's specific community service projects include the handicapped craft sales and the essay contest for high school students, both of which will be described later in this issue of the NEWS, the development of homemaker services, the eye-screening program for pre-school children, and the presentation of the Volunteer Health Service Award.

In addition, the Iowa Medical Society has called upon all doctors' wives to help achieve the objectives of its legislative program. We must not fail our physician husbands. I urge each of you to heed the request of our legislative team, Mrs. Howard Ellis and Mrs. Dean King, about making "Operation Coffee Cup" your number-one project.

Won't those of you who aren't yet members of the Auxiliary decide right now to become members? We want you and we need you! The dues are $3, from July 1, 1961, to March 1, 1962, payable to the state treasurer, Mrs. John Matheson, 4321 California Drive, Des Moines 12.

—MRS. BENJ. F. KILGORE
President

REVISION OF BY-LAWS

The State Auxiliary's By-Laws are being revised for acceptance at the 1962 Annual Meeting. Anyone who wishes to suggest something that she thinks should be added or changed is asked to get in touch with Mrs. R. E. Hines, 3525 Witmer Parkway, Des Moines 10.

1962 ESSAY CONTEST

With school well underway, the time has come to think about our essay contest. This year, let's get the necessary materials to the schools early enough so that we can be sure that pupils will be made aware of its importance and will work up a little competitive spirit.

The topics for the contest are "The Advantages of Private Medical Care" and "The Advantages of the American Free Enterprise System Over Communism." Don't you think them quite appropriate to our present world situation? For many years, as doctors' wives, we have been trying our best to combat the threat of socialism to medicine, and here we have a very practical means of furthering our cause.

There is no better way to advance our crusade than through the minds of our young people. We must encourage these youngsters to explore the situation and come up with their own ideas. The financial rewards are quite attractive. At the national level, the first prize is $1,000; the second prize is $500; the third prize is $250; and there are four additional prizes of $100 each and seven prizes of $75.

The only way for us to get high school students started upon the study of one or the other of these topics is through our local school officials. If the principals can be persuaded to discuss the rules and deadlines for the essay contest with their pupils, we'll be well on our way!

Bulletins have been ordered from the national sponsor, the American Association of Physicians and Surgeons, and each essay-contest chairman and each Auxiliary secretary should be receiving them soon. Please don't delay in getting them to your schools! The contest starts on January 1 and ends on March 1, 1962.

Don't forget to appoint county judges for your essay contest, according to the rules that appear on the back of the bulletin that you receive.

This is such a worthwhile project that we can't afford to neglect it. If we get considerable numbers of high school students to begin thinking about this matter, and if we go on spreading the word about the advantages of private medical care and of the

free enterprise system, we shall have accomplished a great deal!

—AVA CLARE HONKE (MRS. E. M.)
State Community Service Chairman

HANDICAPPED CRAFT SALES

Clay County got off to a head start again with the Handicapped Craft Sale at the fair in Spencer, September 11-17. As we go to press, we don't have the results, but according to Mary Edington the committee was intent upon exceeding last year's receipts, which totaled $1,042.50.

Here are the dates of the sales that have been or still are to be held in 1961:

Waterloo, Mrs. Robert Buckles, chairman, April 7-8.
Fort Dodge, Mrs. C. H. Coughlan, chairman, May 11-13.
Clay, Mrs. F. D. Edington, chairman, Sept. 11-17.
Des Moines, Mrs. N. W. Irving, Jr., chairman, Oct. 2-7.
Dubuque, Mrs. T. J. Benda, chairman, Oct. 23-25.
Sioux City, Mrs. E. D. Ericksen, chairman, Nov. 13-15.

Sioux City hit the high mark for 1960, with a total of $1,184.67 in receipts. Since this is a project for the less fortunate, we can be proud of all that we accomplish, whether large or small. As you know, all profits go directly to the makers of the articles. There's one point in which we can't take pride, however. Only a handful of the 41 local Auxiliaries in the state participate in this charitable work. Does anyone have any ideas on how to increase participation?

FUTURE NURSES AND HEALTH CAREER CLUBS CONFERENCE

The annual conference for Future Nurses and Health Career Clubs will be held at Broadlawns-Polk County Hospital, in Des Moines, on October 20. This state meeting of high school clubs is sponsored by the Woman's Auxiliary each fall, in cooperation with one or more hospitals.

Representatives from all such clubs in Iowa are invited to attend. Programs will be mailed out shortly after October 1.

Auxiliary sponsors, please alert your club president about the earlier-than-usual date. The meeting is being held in mid-autumn in the hope that good weather will make possible an especially large attendance.

COMMUNITY HEALTH SERVICE AWARD

Be sure to keep your eyes open for candidates from your community for "Woman of the Year." After the first of January, letters will be sent out explaining the award for volunteer community health service. Perhaps the lady living just next door to you may deserve this recognition.

COUNTY AUXILIARIES

Black Hawk

The Northeast Iowa Clinical Conference is to be held in Waterloo on Thursday, October 12, and doctors' wives are being extended a special invitation. Registration and meetings are to be held at the Masonic Temple. As a special feature for the women, there will be a luncheon and style show at the Sunnyside Country Club at 1:00 p.m. They will also be interested in the dinner and dance at the Elks Club which is to conclude the day's activities.

The Black Hawk County Auxiliary promises you a warm welcome.

Polk

The Polk County Auxiliary held its spring breakfast on May 12 in the Cloud Room at the Des Moines airport. Mrs. N. W. Irving, Jr. conducted a very impressive installation ceremony for the new officers. A Hawaiian theme was carried out in decorations and food, and the paper leis that the members were given on their arrival helped make the breakfast table colorful. The members had been asked to come dressed either as natives or as tourists, and Mrs. F. C. Coleman won first prize for her impersonation of the much-encumbered tourist. Mrs. Mary Frances Boyd, a former professor of romance languages at Drake University and a veteran traveler, talked on Hawaii. With all her enthusiasm and abundance of humor, she made everyone present anxious to board one of the noisy planes on the runways outside and head straight, non-stop for the Islands.

The new officers held their first board meeting on June 6 at the home of the new president, Mrs. D. W. Kast, to start plans for the year ahead. The first fall executive board meeting was held at the home of Mrs. F. M. Burgeson on September 12. Further board meetings are scheduled for October 3 and 31.

The annual fall party sponsored jointly by the Polk County Medical Society and its Auxiliary was held at the Wakonda Club, in Des Moines, on September 30. It was an evening of fun and fellowship which will be described in greater detail in the November NEWS.

Mrs. Irving is the local chairman for the Handicapped Crafts Sale that is to be held in the fifth-floor foyer at Younkers, Des Moines, from Monday October 2 through Saturday October 7. This sale is held each year under the joint sponsorship of the Polk County Auxiliary and the Iowa Society for Crippled Children and Adults, the Easter Seal Society.

HOW WILL YOUR DUES BE SPENT?

Annual Budget for 1961-1962
Woman's Auxiliary to the Iowa Medical Society

ESTIMATED RECEIPTS

1,125 Members	$3,375.00
Cash on hand	500.00
	$3,875.00

ESTIMATED EXPENSE

I. *Dues to National Auxiliary*		$1,125.00
II. *Administration*		
Yearbook	$200.00	
Postage and Supplies	75.00	
Telephone	50.00	
Two Auditing Fees	20.00	
Bonding Two Treasurers (State and HELF)	10.00	
President's Expense	425.00	
President-elect's Expense	200.00	
Immediate Past President's Expense (A.M.A. Convention)	150.00	
Executive Board Meetings—(Mileage—4 cents a mile)	100.00	
Luncheons for Three Board of Directors Meetings	75.00	
Total Administration Expense		1,305.00
III. *Activities*		
Convention (Annual State Meeting)	$500.00	
Delegates to A.M.A. Convention	60.00	
Bylaws Committee	25.00	
Community Service Committee	35.00	
Woman of the Year Award	$10.00	
Iowa Council for Better Education	15.00	
General Expense	10.00	
Courtesy Committee	90.00	
Gifts	50.00	
Corsages for President and President-Elect	15.00	
Memorials AMEF and HELF	15.00	
General Expense	10.00	
Health Careers Recruitment Committee	100.00	
Iowa Nursing Careers Committee, dues	70.00	
F. N. C. Convention	30.00	
Health Educational Loan Fund Expense	35.00	
Legislative Committee	100.00	
Membership and Organization	300.00	
Chairman	80.00	
District Councilors (11)	220.00	
Nominating Committee	30.00	
Rural Health Committee	45.00	
Other Committees	50.00	
Miscellaneous Expense	75.00	
Total Activities Expense		1,445.00
Total Expense		$3,875.00

We have tried to distribute the available money in proportion to the disbursements of previous committees. In other words, we have added as much as we could to the allocations for officers and committee chairmen whose expenses have been greater during the past year than they had previously been.

Since our membership is about 1,125, the dues collected in Iowa total $3 375. But $1 for each member goes for National Auxiliary dues, leaving us $2 per member for use within the state. Thus our working basis is $2,250 per year.

—MRS. E. A. VORISEK
Finance Chairman

HELP!

The editor of your WOMAN'S AUXILIARY NEWS asks your help in making this publication as interesting as possible to all readers. As an Auxiliary member, won't you please ask your president or publicity chairman to report all activities and projects carried on within your organization, or sponsored by it in your community? If you found it interesting, rest assured that it will interest others and perhaps help them in making their own plans.

Please send clippings or reports to Mrs. Herbert Shulman, 101 Martin Road, Waterloo.

A PRAYER FOR BUSY AUXILIARY MEMBERS

Slow me down, Lord. I'm going too fast.
I can't see my brother when he's walking past.
I miss a lot of good things day by day.
I don't know a blessing when it comes my way.

—Anonymous

COUNTY MEDICAL SOCIETY OFFICERS

COUNTY	PRESIDENT	SECRETARY	DEPUTY COUNCILOR
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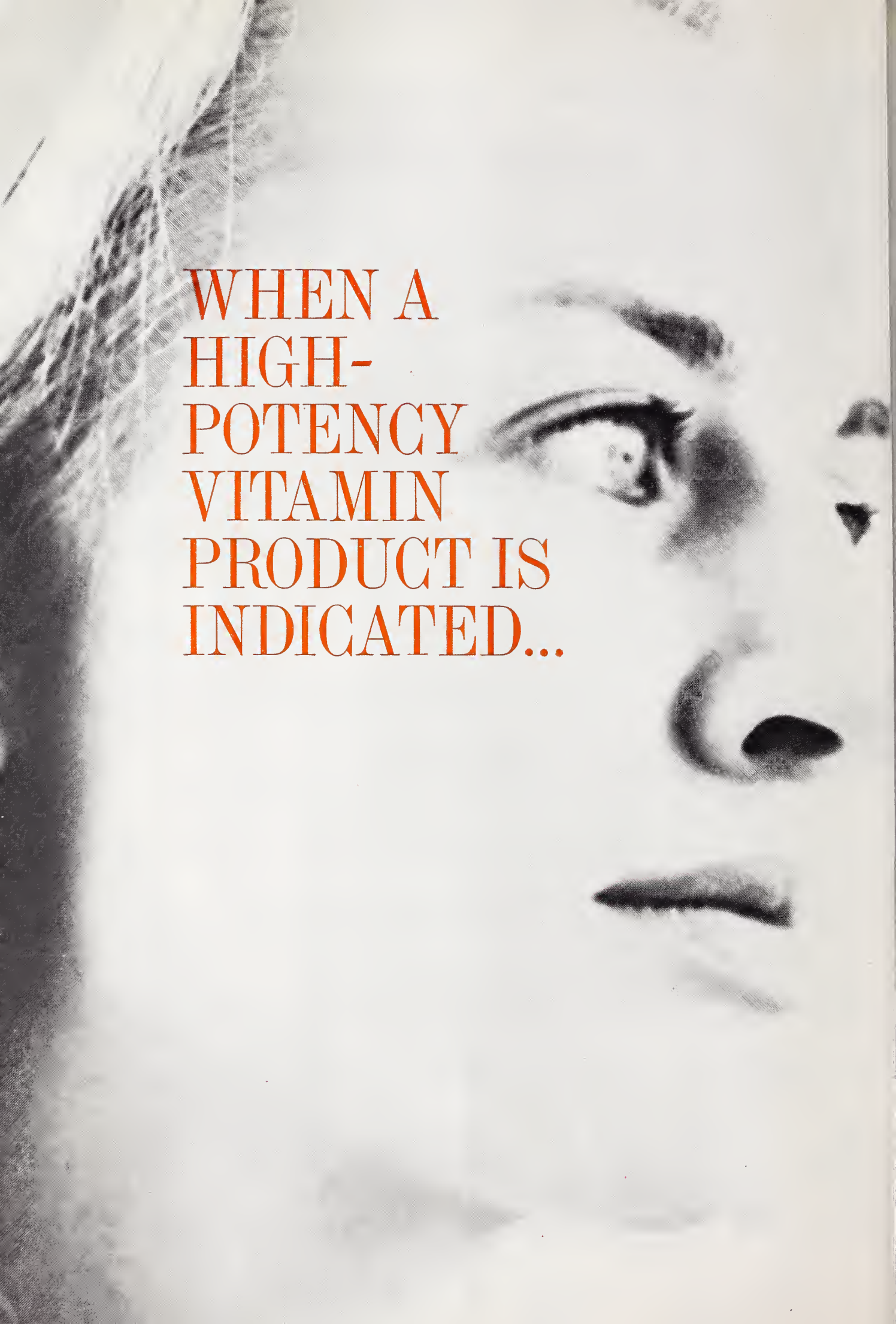
JOURNAL of The IOWA MEDICAL SOCIETY

IN THIS ISSUE:

NOVEMBER, 1961


WHEN A
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The JOURNAL of THE IOWA MEDICAL SOCIETY

Vol. LI NOVEMBER, 1961 No. 11

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The AMA and the Nursing Profession

VERONICA L. CONLEY, Ph.D

CHICAGO, ILLINOIS

The American Medical Association is interested in maintaining the best possible liaison with the nursing profession in the interests of good patient care. If physicians and nurses are to utilize their joint potential to the fullest in the preventive, curative and restorative aspects of health care, a harmonious, collaborative relationship is essential. This cooperation is essential not only for the individual physician and nurse at the patient's bedside, but also for those two individuals as representatives of organizations. Nurses and physicians must not only have a common denominator of interest in the patient and a common body of knowledge, but also must have a deep appreciation of and respect for each other as allies working toward the same goals.

NEWLY-ESTABLISHED AMA COMMITTEE

As a means of strengthening medical-nursing professional relationships, the AMA Board of Trustees has for several years maintained a committee on nursing, now called the Committee for Liaison with National Nursing Organizations. Dr. Charles Hudson, of Cleveland, was appointed acting chairman of this Committee to serve in place of the late Dr. Cleon Nafe, its former chairman. Other members of the Committee are Dr. Elias Faison, of North Carolina, Dr. Clarence Benage, of Kansas, Dr. Elmer Weigel, of New Jersey, and Dr. Arthur Kirchner, of California.* In order to provide a headquarters staff for this Liaison Committee, to strengthen and expand its activities, a Department of Nursing was established during the past year.

The Committee hopes to accomplish the following objectives:

1. To strengthen medical-nursing profession liaison at the national, state and local levels
2. To study and report on practices and trends in nursing as they pertain to the medical profession
3. To stimulate, initiate and, where feasible, support research in areas pertinent to the nurse-physician relationship in professional practice
4. To serve in an advisory capacity to the medical profession on matters of interrelationships between the two professions
5. To provide support and assistance to the nursing profession in its efforts to maintain high standards
6. To encourage physicians to respond to requests from nurses to serve on nursing-school faculties.

The Committee proposes to accomplish these objectives through a program based on four major activities: (1) Liaison, (2) Education, (3) Consultation, and (4) Research.

A CONTINUING TASK FOR MEDICAL AUXILIARIES

Members of the Auxiliary, I know, are interested in learning how they can help achieve the objective of improved liaison between the medical and nursing professions. In preparing for this talk, I asked one of the leaders in nursing whether there were any points she felt I should stress. She replied: "The Medical Auxiliaries all over the country do such a tremendous job in recruitment for nursing and all health careers that I would want them to know how sincerely appreciative we are of their fine work. Their sponsorship of Future Nurses Clubs and/or Health Careers Clubs, as well as their provision of nursing scholarships, are invaluable to the nursing profession."

The most obvious role of the Woman's Auxiliary is in helping the nursing profession, through recruitment activities, to maintain the quality and quantity of its membership. Few facts are so well publicized as the existence of a shortage of professional nurses. It is not so well known that the nursing profession has made its greatest gain in members during the past 20 years. There are now over one-half million active professional nurses, making nursing one of the largest of the health professions. The need for nurses is expanding at an alarming rate in hospitals, in the community and in industry. We urge that recruitment be accelerated for all three basic programs leading to professional nurse status—the diploma program, the associate degree (two-year) program and the baccalaureate program. We urge something else—namely that young women contemplating nursing be clearly told the characteristics of each of those three programs so that they may select the program which will utilize their potentialities to the utmost. A wise selection will help to keep that student in nursing and ultimately to provide the best patient care.

Your efforts in relation to scholarships are es-

Dr. Conley, the director of the AMA Department of Nursing, gave this address in New York City on June 27, at the annual convention of the Woman's Auxiliary to the AMA.

* Since this paper was presented, Dr. Hudson has left the Committee, and Dr. Kirchner has succeeded him as chairman. There are now three new members—Dr. W. Benson Harer, of Pennsylvania, Dr. Charles L. Leedham, of Ohio, and Dr. William R. Willard, of Kentucky. Dr. Leonard W. Larson, of North Dakota, president, Dr. George M. Fister, of Utah, president-elect, and Dr. Hugh M. Hussey, of the District of Columbia, chairman of the Board of Trustees of the AMA, are ex-officio members of the Committee.

sential. Nursing education is becoming increasingly expensive. College programs are just as costly, as regards tuition, as is any other area of study, and scholarships are badly needed. It is from these programs that future teachers of nurses are expected to come. There is a critical shortage of teachers at present. In behalf of the Liaison Committee, I urge your continued efforts in recruitment.

The second major role that the Woman's Auxiliary can assume is that of providing moral support for the nursing profession. Nursing, just like medicine, has been subjected during the past decade to considerable criticism and comment by allied professions, by consumers of nursing service and by the press. A major source of the criticism is the lack of understanding of what constitutes medicine and nursing in this last half of the twentieth century. Today's society is quite unlike the one that existed 50 years ago, and societies shape their institutions. The humanitarian aspects of medicine and nursing have not changed, but their functions have.

NURSING, LIKE MEDICINE, HAS CHANGED

Not too many years ago, the public quite accurately pictured the average physician as watching through the night at the patient's bedside in the family home. He was there to give what he could of himself to all members of the family for as long as he was needed. His diagnostic measures were crude, his effective drugs few, and his contribution was largely one of comfort, watchful waiting and prayer.

In the past decade this traditional relationship has been altered. The physician is no longer monarch of his patient's medical problems. Where once he assumed full responsibility, seven to 10 other people now perform one or more professional services each for his hospitalized patient. The physician has been replaced at the patient's bedside, for a good part of the time, by others.

The evolution of the nurse's role has taken a similar direction. The complex hospital environment and the changes that have taken place in medical care have forced her away from the patient's bedside. The physician has had to turn over to her many of his former functions because he no longer has the time to carry them out. At one time, nurses did not take temperatures or blood pressures, but now they perform those tasks and, in addition, administer intravenous infusions and even blood transfusions. The nurse has had to learn to operate some of the most fantastically complex equipment man has ever devised. More than ever we depend on her clinical judgment, her vigilance and her educational preparedness in life-and-death matters. But the professional nurse has, in turn, had to delegate some of her simpler and more routine tasks to practical nurses and aides. She not only must be a more skilled practitioner than ever before but must know how to direct and teach the many other hospital personnel who now minister to the patient. All of this is essential in twentieth century patient care. The physician has had to become less personal and more scientific, and so has the nurse.

We must help to see to it—you and I—that these two professions employ self-study and research in an effort to strike a happy balance between the scientific and the personal, so that they may continue to strive for the best patient care the world has ever known.

Give to the American Medical Education Foundation

It has taken the individual contributions of many physicians to achieve the more than $10,000,000 already channeled through AMEF to the nation's medical schools. The challenge, however, is still present, and the unified support of all physicians is needed more than ever.

Additional funds are needed in every area of medical training, in the state schools as well as those that are privately endowed. Since an increase of 50 per cent will be needed in the number of graduating M.D.'s by 1975, the schools are being pressed to expand. At the same time, they are required to keep abreast with the rapid growth of medical technology. Students and prospective students are faced with rising costs and a general lack of available loan funds for medical students. The support we give to the AMEF program is the most direct assistance we can provide in meeting these problems of medical education. The funds that AMEF gives to medical schools are unrestricted; the dean of each college may use them at his discretion.

The AMEF is beginning its second decade as a very important part of organized medicine's effort to provide financial support to medical education and to keep it free of federal control. Let's give it our unanimous endorsement by making our contributions to it now!

Scientific Articles

The Physiology of the Pancreas

DANIEL B. STONE, M.B., and JUDITH GRAHAM, M.D.
IOWA CITY

THE PANCREAS CONSISTS of the acini and the islets of Langerhans. The islets of the human pancreas contain three types of cells. The beta cells secrete insulin. The alpha cells probably secrete glucagon. Despite the fact that the third type of cell, the delta cell, was first described 30 years ago,[1] its functions remain unknown.

The work of Homans, eight years before the discovery of insulin, suggested that the beta cells were the source of the antidiabetic hormone in the cat.[2] Alloxan produces diabetes through the destruction of the beta cell. Nearly all diabetics have a reduction in the total mass of beta cells without any definite alteration in the total mass of alpha cells.[3-5] Recent bioassay and histologic studies show that pancreatic insulin content closely parallels the number of beta cell granules. The beta cell granules probably represent stored insulin. Perfusion of the isolated pancreas with hyperglycemic blood stimulates the release of insulin into the pancreatic vein, and partial degranulation of the beta cells follows. Nobody knows the mechanism by which the beta cell granule releases its insulin, or the manner in which the released insulin enters the blood stream.

In 1923, pancreatic extracts were found to cause hyperglycemia.[6] Later, the hyperglycemic factor, glucagon, was characterized and crystallized. The accumulated evidence suggests that the pancreatic alpha cells probably manufacture and store glucagon, perhaps as granules. There is no evidence that any specific alpha cell stains identify glucagon. Selective destruction of the alpha cells might inhibit glucagon manufacture. Results of cytotoxic alterations of the alpha cells, however, are variable and contradictory because of other effects of the toxic agents.[7-11] The evidence certainly suggests, though it does not prove, that the alpha cells make glucagon.

Since one of the aims of the endocrinologist is to equate hormonal action with enzymatic activity and biochemical changes, it is necessary to review intermediary metabolism before considering the actions of insulin and glucagon. After describing cellular metabolism, we shall discuss metabolism in certain tissues, the changes after insulin administration and in diabetes mellitus, and lastly, the actions of insulin and glucagon.

Both insulin and glucagon influence lipid and protein as well as carbohydrate metabolism. Although the most striking metabolic lesion in diabetes mellitus is hyperglycemia, biochemical aberrations of fat and protein, though less easily demonstrable, may be more important. Clearly, diabetes is a general disturbance of metabolism, with the changes in lipids sometimes inconspicuous and sometimes dominant—for instance, in patients with so-called idiopathic hyperlipemia and mild diabetes. Recently, the lipid derangements of diabetes mellitus have come into sharp focus.

CARBOHYDRATES

Ingested carbohydrate is split into monosaccharides in the gut and then absorbed, both actively and passively. Figure 1 shows the general scheme of intermediary metabolism and the interplay of fat, protein and glucose, simplified for purposes of demonstration. Glucose must first enter the cell. Once inside the cell, it is phosphorylated via the hexokinase reaction to glucose-6-phosphate. Thereafter, several pathways are open to it. Free glucose may be formed from glucose-6-phosphate, the reaction catalyzed by glucose-6-phosphatase. This occurs chiefly in the liver; the glucose-6-phosphate may be derived from glycogen (by *glycolysis*), or from protein or fat

Dr. Stone is an associate professor of internal medicine at S.U.I., and Dr. Graham is an N.I.H. trainee in diabetes and instructor in internal medicine at S.U.I.

precursors (by *gluconeogenesis*). A second route open to glucose-6-phosphate is glycogen synthesis.

The two major routes taken by glucose-6-phosphate, however, lead into the maze that proceeds to the final common pathway of all foods, the Krebs cycle. The first of the two roads to the Krebs cycle is the Embden-Meyerhof pathway of anaerobic glycolysis. The conversion of one mole of glucose to two moles of pyruvate via anaerobic glycolysis requires two moles of adenosine triphosphate (ATP) for phosphorylation of the hexose. The hexose phosphate subsequently breaks down into two three-carbon compounds. The metabolism of these two three-carbon compounds to pyruvate yields four moles of ATP and two moles of reduced diphosphopyridine nucleotide (DPNH). These two moles of DPNH can then be oxidized to DPN, yielding six additional moles of ATP. If oxygen is absent (as in working muscle), the hydrogen of DPNH can be used to reduce pyruvate to lactate. Under anaerobic conditions, the overall reaction is therefore:

$$\text{Glucose} + 2\ \text{ATP} \rightarrow 2\ \text{lactate} + 4\ \text{ATP}$$

For each mole of glucose metabolized to lactate there is a net gain of 2 moles of ATP. If the reaction stops at pyruvate, the overall reaction is:

$$\text{Glucose} + 2\ \text{ATP} + 2\ \text{DPN} \rightarrow 2\ \text{pyruvate} + 4\ \text{ATP} + 2\ \text{DPNH}$$
$$2\ \text{DPNH} + 6\ \text{ADP} \rightarrow 2\ \text{DPN} + 6\ \text{ATP}$$

Thus, in this case, there is a net gain of eight moles of ATP. ATP represents stored energy, each mole being equivalent to about 8,000 calories. Energy produced in these reactions is only a small fraction of the total available from glucose, but it is available in the absence of oxygen. Consequently, the process is called "anaerobic glycolysis."

The second route to the Krebs cycle, found chiefly in tissues actively synthesizing fatty acids

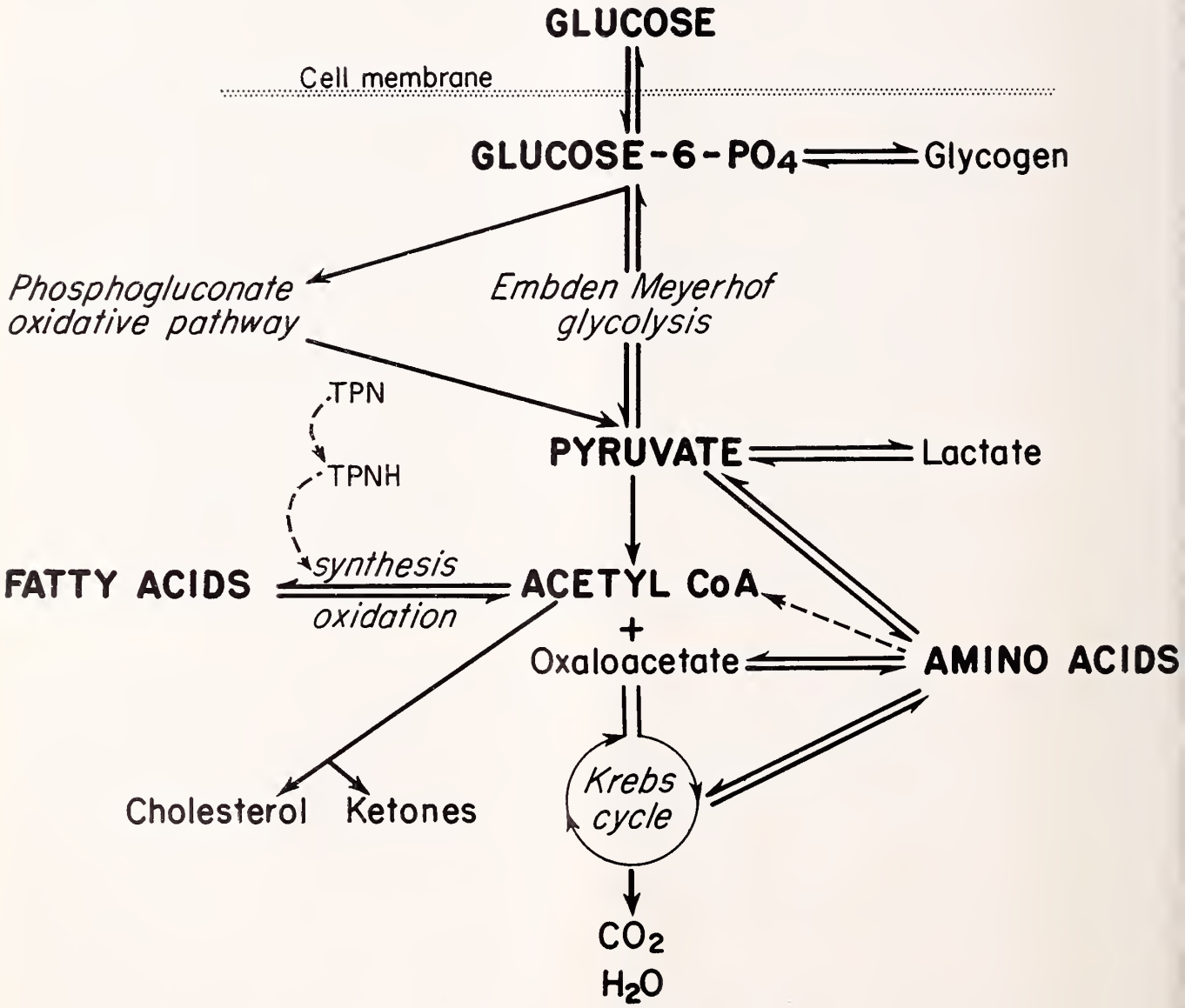

Figure 1. Outline of intermediary metabolism.

such as liver and adipose tissue, is the phosphogluconate-oxidative pathway (also called the pentose phosphate pathway or hexose-monophosphate shunt). The importance of this route is that for each mole of glucose using it, two moles of reduced triphosphopyridine nucleotide (TPNH) are formed. The overall reaction is:

3 glucose-6-phosphate + 6 TPN → 3 CO_2 + 6 TPNH + 6 H + 2 fructose-6-phosphate + 1 glyceraldehyde-3-phosphate

The supply of TPNH is perhaps the controlling factor in lipogenesis, as we shall point out later, and thus may be particularly important in the diabetic's metabolism.

The Krebs cycle is analogous to a merry-go-round, with 2- and 3-carbon fragments of carbohydrate, fat and protein entering and leaving at several points. During the spin, the lower intermediates of these three basic groups of foods blend to become indistinguishable, and ATP spins out, along with CO_2 and water. With each revolution, 15 moles of ATP are generated from each mole of pyruvate. Less is known about the Krebs cycle than about anaerobic glycolysis, but it is clear that the oxidation of pyruvate yields much more energy than does glycolysis. Most of the energy derived from glucose metabolism comes from the oxidation of pyruvate in the Krebs cycle. Since one mole of glucose yields two moles of pyruvate, the total ATP gain from a single mole of glucose is 2 + 6 + (15 × 2) = 38 moles of ATP, and 38 × 8,000 = 304,000 calories. This is about 45 per cent of the theoretical total caloric yield from the transformation of one mole of glucose to CO_2 and water.

LIPIDS

Diabetes might have been regarded as a disease of fat, rather than of carbohydrate metabolism, if blood fats could have been measured before blood sugar. Diabetics show ketone-body accumulation, decreased lipogenesis, increased dependence on fatty acid breakdown for energy, and enhanced cholesterol synthesis. Lipogenesis is important to the normal body. Only 3 per cent of ingested glucose is stored as glycogen and 90 per cent as fat, so that the body's carbohydrate stores will suffice for less than one day of fasting. Adipose tissue has been found to be a tremendously active organ, accounting for the bulk of lipogenesis, rather than the inert storage depot that it was formerly thought to be.

Figure 2 is an enlargement of a part of Figure 1, showing certain details of lipogenesis and fatty acid oxidation (breakdown), the defects in diabetes and the development of diabetic ketosis. Most of the fatty acids synthesized within the cell derive their carbon from the common catabolite of foodstuffs, acetyl CoA. The initial reaction of lipogenesis involves the condensation of two moles of acetyl CoA to form one of acetoacetyl CoA, which is then reduced, dehydrated and again reduced to form butyryl CoA (the first true activated fatty acid). Since this reduction is probably the controlling reaction of lipogenesis, it should be emphasized that TPNH serves as the hydrogen donor in the reduction of crotonyl CoA, and so a lack

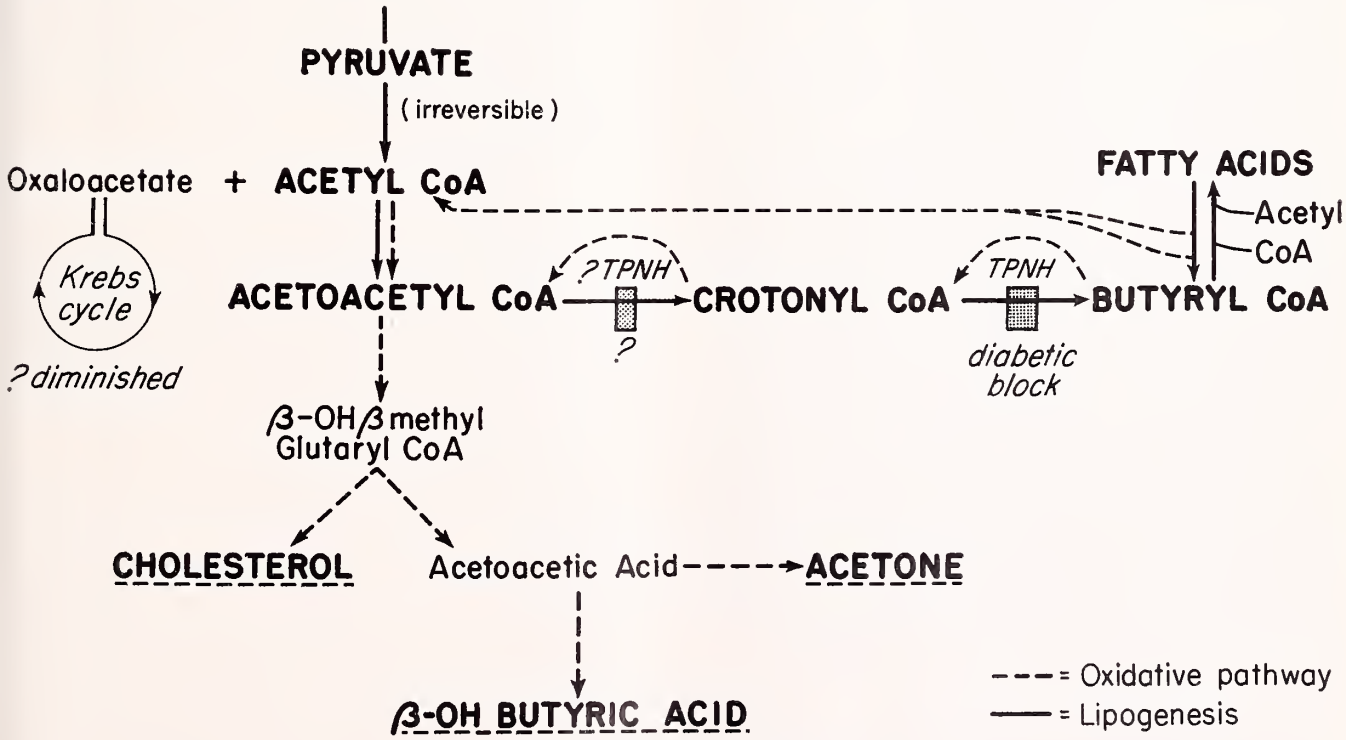

Figure 2. Fat metabolism in diabetes.

of TPNH may well be the limiting factor in lipogenesis. As we have seen, TPNH is derived from the phosphogluconate-oxidative pathway, most active in tissues that synthesize fatty acids. By repeated condensations with acetyl CoA and reduction, a long-chain fatty acid is ultimately formed. It can then form free (non-esterified) fatty acid, combine with glycerol to form triglycerides, or form esters with cholesterol.

There is now good evidence to show that a lack of TPNH is probably responsible for the failure of the diabetic to synthesize fat, and thus that the exact location of the lipogenic block is at the site of formation of butyryl CoA. Insulin stimulates both the phosphogluconate-oxidative process and lipogenesis.

Oxidation of fat produces energy. Triglycerides are hydrolyzed to free fatty acids, which are converted to the fatty acid: CoA form and then broken down, one 2-carbon unit at a time. At each step in this breakdown, one mole each of DPNH, flavin adenine dinucleotide (FADH) and acetyl CoA are formed. The acetyl CoA enters the Krebs cycle, yielding ATP. The FADH and DPNH are oxidized, yielding more ATP. Lipogenesis and fatty acid oxidation occur at different points within the cell, and require different enzyme systems. TPNH is *not* required in fatty acid oxidation. The diabetic oxidizes fat at an increased rate, apparently because of a lack of glucose utilization. The exact relationship between the two abnormalities is not known.

Figure 2 also brings together the present knowledge concerning the production of *diabetic ketosis*. There is over-production of acetyl CoA because of increased fatty acid oxidation. Surplus acetyl CoA cannot be shunted back to pyruvate, for this reaction is irreversible. The two usual processes of acetyl CoA disposal are said to be impaired: the conversion of acetyl CoA to fat is blocked in the diabetic; the supply of oxaloacetate for the Krebs cycle is said to be decreased so that oxidation of acetyl CoA by this route is impaired. There is dispute, however, about the failure of oxaloacetate supply. The results of direct measurement of Krebs cycle intermediates and of the administration of oxaloacetate to ketotic laboratory animals and diabetic human beings are variable. Krebs[12] reviewed these aspects of ketosis and concluded: "There is no evidence of under-utilization of acetyl coenzyme A in the ketotic organism, in the sense of the reduced rate of the tricarboxylic acid cycle. The capacity of the body as a whole to burn acetate is never fully used in a resting organism." Nevertheless, the excess acetyl CoA *does* accumulate and *is* converted to acetoacetate and ketones, or to cholesterol.

DIABETIC KETOACIDOSIS

Since insulin facilitates the utilization of glucose by certain tissues, especially adipose tissue and muscle, relative lack of insulin action results in hyperglycemia. Furthermore, increased gluconeogenesis aggravates this hyperglycemia. The accelerated gluconeogenesis can be thought of as an attempt by the liver to compensate for a failure of peripheral glucose utilization, whether due to insulin lack (diabetes mellitus) or glucose lack (starvation). The resultant hyperglycemia tends to correct for the defective utilization, but only in part. In the absence of ketoacidosis, it would appear that such a compensation does occur in the diabetic, although it is generally assumed that this is associated with states of partial, rather than complete, insulin deficiency. In terms of overall carbohydrate balance, a major part of the problem that faces the diabetic stems from the occurrence of massive glycosuria when hyperglycemia surpasses the renal threshold for glucose. The effectiveness of hyperglycemia is wasted by the rapidly mounting urinary loss. Massive glycosuria provokes osmotic diuresis.

Excessive hepatic gluconeogenesis entails the simultaneous metabolism of fatty acids and protein. Fatty acids provide the necessary hydrogen for the reduction of pyruvate to glucose. Protein contributes carbon that ultimately becomes the pyruvate itself. Diabetics who lose large quantities of glucose in their urine oxidize a proportional amount of fatty acid, generating large numbers of ketone bodies. When the production of ketone bodies surpasses the ability of the body to use them, they accumulate in the blood. Since their renal threshold is very low, an increasing amount is lost in the urine. Having relatively low pK values, they are excreted predominantly as anions. The cations, sodium and potassium, are excreted with them. This loss of base aggravates not only the osmotic diuresis induced by the glycosuria, but also the metabolic acidosis produced by the ketones.

METABOLISM IN INDIVIDUAL TISSUES

It is important to recognize the metabolic individuality of different tissues, for hormones differ in their actions in various tissues. Tissue responsiveness and metabolic individuality depend upon enzymatic content. We shall discuss metabolism in three individual tissues: muscle, adipose tissue and liver.

Metabolism in Muscle: The main function of muscle is the storage of chemical energy and its transformation into mechanical energy. Muscle metabolism is mainly glycolytic and relatively simple. In muscle tissue, there is neither phosphogluconate-oxidative pathway activity nor lipogenesis. These two absences may obviously be related, since TPNH production by the phosphogluconate-oxidative pathway is essential for adequate lipogenesis. Muscle cells lack glucose-6-phosphatase, and consequently muscle does not liberate glucose into the blood. Skeletal muscle

has three main metabolic activities: (a) Glucose enters muscle cells and may be stored as glycogen. (b) When required, glycogen may be mobilized and converted to mechanical energy via anaerobic glycolysis and the Krebs cycle. (c) Free fatty acids (and ketone bodies) are also used as fuel for muscular exercise.

Metabolism in Adipose Tissue: Formerly, adipose tissue was regarded as a static storage depot, but it is now recognized to be an extremely active metabolic organ. In adipose tissue, intermediary metabolism is centered around fatty-acid synthesis, release and oxidation. Adipose tissue lipid is synthesized from both glucose and triglycerides. Glucose is now known to be an important fatty acid precursor, providing necessary acetyl CoA. Some 50 per cent of glucose is metabolized via the phosphogluconate-oxidative pathway in order to generate the required TPNH. Triglyceride may be incorporated into adipose tissue directly or may undergo hydrolysis and be resynthesized. Catabolism in adipose tissue involves the breakdown of fat into fatty acids, which are then oxidized. Adipose tissue releases fatty acids during carbohydrate deprivation, either during fasting or in diabetes mellitus.

Metabolism in the Liver: The liver can store carbohydrate by forming glycogen (*glycogenesis*), and it can release this glycogen when required (*glycogenolysis*). The liver "buffers" the blood sugar level and supplies nearly all the endogenous sugar in the blood, insignificant amounts coming from the kidney. All tissues are able to take glucose from the blood and convert it to glucose-6-phosphate, but in tissues other than liver and kidney, this reaction is irreversible. Only liver and kidney can supply glucose to the blood, for only they contain glucose-6-phosphatase. The liver can also form glucose from non-carbohydrates such as amino acid and fat, and from intermediates such as glycerol and lactate (*gluconeogenesis*). The rate of gluconeogenesis from amino acid or fat depends upon the availability of carbohydrate. Ample carbohydrate spares protein and fat, whereas relative carbohydrate lack (or underutilization) encourages the breakdown of protein and fat. The liver also performs dual functions in fat and protein metabolism, supporting anabolism or catabolism, according to need.

EFFECTS OF INSULIN AND OF DIABETES ON INDIVIDUAL TISSUES

Muscle: The influence of insulin on muscle *in vivo* is related to the blood sugar level. It is clear that insulin enhances the uptake of glucose and increases glycogen stores in skeletal muscle. If, as a result of this action, the blood sugar is reduced, these insulin effects are modified by epinephrine output from the adrenal medulla. Epinephrine causes glycogenolysis in muscle. Muscles of diabetic animals or man show decreased glycogen stores, diminished glycolysis, increased ketone body and free fatty acid utilization and increased protein breakdown.

Adipose Tissue: In adipose tissue, insulin increases glucose uptake and utilization, accelerates lipogenesis, and decreases fatty acid oxidation and free fatty acid release. Diabetic adipose tissue shows decreased glucose uptake and utilization, increased fatty acid oxidation and (as with fasting) increased free fatty acid release.

Liver: The action of insulin on the liver *in vivo* is complicated and controversial. Diabetic liver is clearly abnormal. It shows decreased glycogen synthesis, increased glycogenolysis, decreased lipogenesis and increased gluconeogenesis. These abnormalities are corrected when insulin is given to the diabetic. This suggests that insulin acts directly on hepatic metabolism. Other evidence, however, tends to refute this conclusion. The effects of insulin on the livers of experimental animals and human diabetics are delayed and may be secondary to peripheral insulin action. Direct effects of insulin have, indeed, been demonstrated on isolated liver preparations *in vitro*. Experiments *in vivo*, both in diabetic humans and in diabetic animals, have given inconsistent results. Insulin in normal animals actually decreases hepatic glycogen. At this time, the direct influence of insulin on the liver appears to be of minor physiologic significance, certainly as regards hepatic glucose uptake and release.

TABLE I

MULTIPLE METABOLIC CHANGES FOLLOWING AN INJECTION OF INSULIN AND OCCURRING WITH DIABETES MELLITUS

	Effects of Insulin	Effects of Diabetes Mellitus
Level of blood sugar	Decreased	Increased
Glycogen stores of skeletal muscle	Increased	Decreased
Plasma lipids:		
Total lipids	Decreased	Often increased
Total cholesterol	Decreased	Often increased
Phospholipids	Decreased	Often increased
Triglycerides	Decreased	Usually increased
Free fatty acids	Decreased	Increased
Fat synthesis	Increased	Decreased
Fatty acid oxidation	Decreased	Increased
"Ketone body" formation	Decreased	Increased
Respiratory quotient	Elevated	Decreased
Protein metabolism	Catabolism inhibited	Catabolism increased
Serum inorganic phosphate	Reduced	Increased
Intracellular potassium	Increased	Reduced

To summarize, we might say that in skeletal muscle, insulin increases glucose uptake and glycogen synthesis. In adipose tissue, insulin not only enhances glucose utilization (both via the Embden-Meyerhof glycolytic pathway and via the phosphogluconate-oxidative pathway), but secondarily accelerates lipogenesis. The evidence suggests that the liver is a minor or secondary site of insulin action, although insulin may change hepatic carbohydrate metabolism. The resolution of the effects of insulin on adipose tissue and muscle, which depends upon the enzymatic individuality of these tissues, explains the multiple metabolic effects of insulin listed in Table 1.

MECHANISM OF ACTION OF INSULIN

Many observations suggest that insulin influences some early process in the chain of glucose metabolism, such as entry into the cell or initial phosphorylation. (a) Insulin accelerates glucose utilization. (b) Insulin does not alter the rate of chemical reactions *in vitro if the cell membranes are destroyed.* (c) Tissues which are least permeable to glucose (such as skeletal muscle and adipose tissues) are most responsive to insulin. Conversely, tissues which have membranes more permeable to glucose transfer (such as liver) respond less to insulin.

Cori *et al.*[13] focused attention upon phosphorylation as one early common path of glucose metabolism. They were unable to show that insulin directly activates the hexokinase reaction. They believed that they had shown, however, that insulin releases hexokinase from anterior pituitary or adrenocortical inhibition, and they postulated the hexokinase enzyme system as the site of insulin action. There are several criticisms of this hypothesis. (a) Insulin has striking effects in both hypophysectomized and adrenalectomized-hypophysectomized animals. (b) The results of Cori's experiments have not been fully confirmed by other investigators. There is, therefore, little support for the hypothesis that insulin acts by releasing hexokinase from inhibition.

The mechanism of insulin action may be found at an even earlier point than that of phosphorylation. Levine and Goldstein[14] showed that insulin enhanced the penetration of galactose in the cells of eviscerated, nephrectomized dogs. Galactose is non-utilizable in these cells. Insulin reduced the concentration of sugar in extracellular fluid, despite the fact that it did not influence its utilization in the cell. Levine *et al.* then developed the "permeability" hypothesis of insulin action, suggesting that insulin acts by transferring glucose from extracellular fluid across the cell membrane into the cell. The findings of these investigators have received ample confirmation through a number of different experimental technics. A reasonable conclusion at this time is that insulin acts by regulating the rate of entry of glucose into peripheral cells. This conclusion allows a unifying concept of the mechanism of action of insulin.

GLUCAGON

Glucagon causes hyperglycemia when injected into animals having adequate stores of liver glycogen. It appears to cause glycogenolysis, but not gluconeogenesis. Starvation, diabetic acidosis, glycogen storage disease, parenchymal liver disease and hepatectomy diminish or abolish the hyperglycemic response. Glucagon either fails to affect glucose utilization in the peripheral tissues, or increases its rate slightly. Glucagon-induced hyperglycemia results in an initial fall and a later rise in the blood level of free fatty acids.

Chronic glucagon administration or continuous infusions cause temporary diabetes-like states in various experimental animals. Despite the fact that large doses of glucagon given to rats caused hyperglycemia and death in apparent acidosis,[15] no *permanent* diabetic state has been reported in either experimental animals or man following glucagon administration.

Glucagon may be injected subcutaneously, intramuscularly or intravenously, and may be used in the treatment of hypoglycemia. Patients respond to intramuscular injections within 10 to 15 minutes. A diabetic's family can be taught to give glucagon, a maneuver that may save the life of the diabetic who is unconscious from insulin hypoglycemia.

We thank Dr. William B. Bean and Dr. Robert C. Hardin for their advice and help.

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The Role and Scope of Neuro-Otology

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If we think of neuro-otology as implying neuro-otolaryngology, then we should define it as the borderline between neurology and otolaryngology. The terms of reference in this interdisciplinary area are shown in Figure 1, and it will be convenient to discuss the various aspects in the order provided by the capital-lettered subheadings there.

(A) Investigation of Neurological Symptoms Referable to the ENT Area

This section covers patients with, for example, a variety of head and neck pains who are referred for neuro-otolaryngological evaluation. A common problem is whether a headache could be attributed to sinusitis. In this respect, and with regard to children, one should remember Goodhill's aphorism: "Headache due to sinusitis is uncommon in children."[61]

Malignancies of the postnasal space frequently present as facial pain—occurring on an average, 10 or 11 months before the correct diagnosis is made.[59] Initially, in these cases, the postnasal space may even appear normal on visualization, and the diagnosis has been made only on a blind biopsy of the fossa of Rosenmüller.

Glossopharyngeal neuralgia may be due to an elongated styloid process,[134] or it may be the first and only evidence of a malignancy of the base of the tongue. In fact, all cases in one small series that was followed up proved eventually to have malignant neoplasms.

(B) Investigation of Voice and Speech Disorders

At S.U.I., this home of speech pathology, it would be rather presumptuous of me to devote more than a passing reference to this topic. However, as Morley *et al.* have said, continuing uncertainties about many of the cerebral processes involved in speech may make speech disorders a difficult diagnostic problem.[103]

Delay in speaking is a common problem in children. Morley and her coworkers found that 40 per cent of such cases are due to deafness, 25 per cent to mental deficiency, 25 per cent to developmental aphasia,[48] and 8 per cent to cerebral palsy. High-tone hearing losses were not associated with any appreciable delay in speaking.[103]

Nasality problems are frequent in children, and the current interdisciplinary approach to their diagnosis is proving fruitful. Calnan's scheme for

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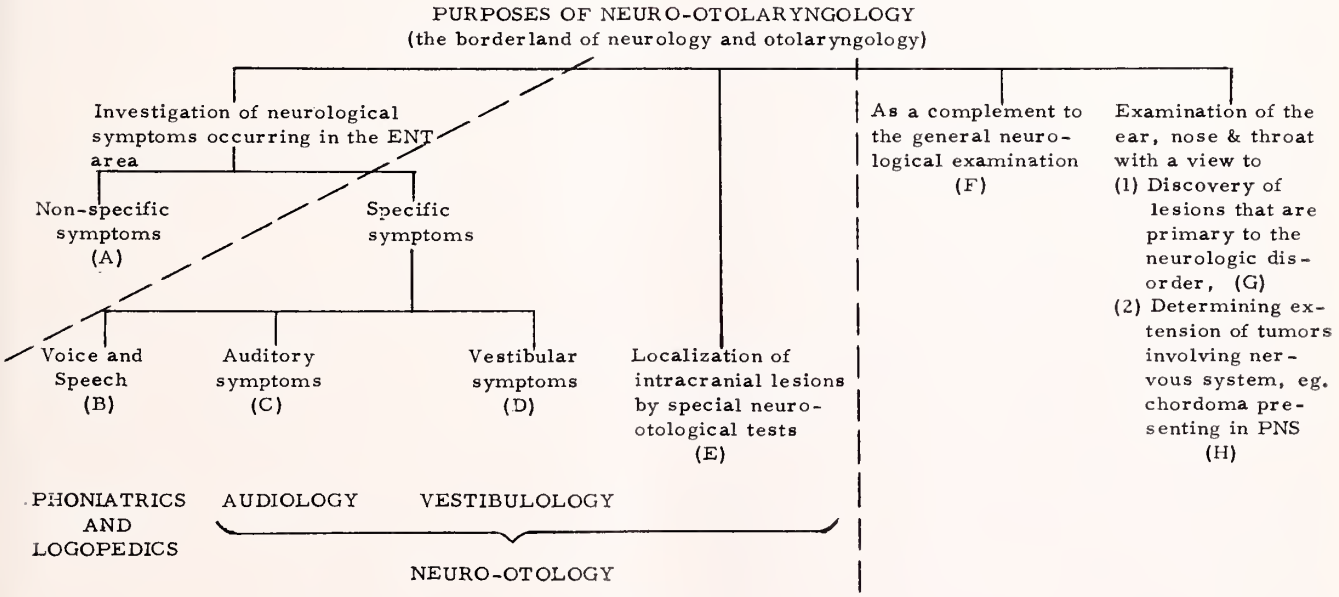

Figure 1.

the investigation of a speech defect with particular reference to nasality[27] is shown in Table 1.

TABLE 1

INVESTIGATION OF A SPEECH DEFECT[27]

A. History
   1. Present complaint
   2. Past: "Milestones," Illnesses
   3. Family: of C.L. and P.; of congenital deformities; of neurological complaints

B. Examination
   1. Inspection
      a. palate, pharynx, tongue
      b. ears and nose
   2. Palpation
   3. Auscultation: speech-articulation; nasal escape; resonance

C. Special Investigations
   1. Audiograms
   2. Manometry
   3. Speech recordings
   4. Neurological
   5. Psychological; intelligence
   6. Social report; school reports
   7. Dental
   8. Lateral radiographs of palate
   9. Cineradiography during speech and swallowing
   10. Endoscopy
   11. Faradic stimulation of palate and pharynx
   12. Measurements of nasopharyngeal isthmus
   13. Biopsy

(c) INVESTIGATION OF AUDITORY SYMPTOMS

The investigation of deafness and tinnitus is one of the basic roles of neuro-otology. Audiometric screening of school children indicates that 120 in every 1,000 have defective hearing, which in nearly 80 per cent of cases is a conductive loss and almost invariably an acquired one.[50] Presumably, many of these conductive deafnesses in children are only temporary, since the prevalence of conductive deafness in adults is of the order of 60 per 1,000 population. By the age of 70 years, over 20 per cent of the population have presbycusis, defined as a hearing loss for speech in excess of 25 decibels.[70]

Congenital hearing deficiencies and hearing defects acquired in infancy usually present as a delay in the development of language. Of children seen for these reasons, Myklebust[105] found that fewer than half the patients suffered from peripheral deafness, 30 per cent were aphasics, 16 per cent were psychogenics and 9 per cent were mental defectives. Congenital peripheral deafness is predominantly of the sensori-neural (i.e., perceptive) type, as opposed to conductive deafness, and is primarily genetically determined.[78] Table 2 lists

TABLE 2

CAUSES OF DEAFNESS IN CHILDREN

(After Falbe-Hansen, 1954, and Johnsen, 1954)

	Per Cent
(A) CONDUCTIVE (78%)—PREDOMINANTLY ACQUIRED	
"Middle ear catarrh"*	33
Post-otitic scarring	22
Cerumen or F.B.	14
Acute or chronic suppurative otitis media	10
(B) PERCEPTIVE (22%)—PREDOMINANTLY CONGENTIAL	
Genetic	9
Due to paranatal asphyxia	2
Related to prematurity	2
Erythroblastosis	1

* This is a loose term which covers not only serous otitis media, but also recurrent otitis media without discharge, auditory tubal block, and, sometimes, even juvenile otosclerosis.

TABLE 3

CAUSES OF CONDUCTIVE DEAFNESS IN ADULTS[70]

Condition	Percentage
1. Chronic suppurative otitis media	32
2. Post-otitic scarring	31
3. Otosclerosis	18
4. Previous radical mastoid operation	5
5. Congenital abnormalities	5
6. Serous otitis media	3
7. Other	6

the principal causes of deafness in children, and Table 3 shows the principal causes of conductive deafness in adults. Until the age of 65 years, or thereabouts, conductive deafness is commoner than sensori-neural deafness.[70]

Recent advances in audiometry have made it possible to obtain considerable information, in a case of deafness, with respect to (a) the locus of the lesion; (b) the degree of the hearing deficit; and (c) the organicity of the defect.

Until a relatively short time ago, determination of the locus of a lesion causing deafness was dependent upon the traditional Rinne tuning-fork test. If the fork is heard better by bone than by air conduction, the deafness is said to be conductive in type, and the cause of the deafness is judged to lie in the external or middle ear. If the fork is heard better by air conduction, then the deafness is termed sensori-neural, and the cause is said to lie in the inner ear or in the auditory pathways. That test is not a quantitative one, but it has been shown to be a valid and reliable indication of conductive deafness.[71] The emergence of combined

air and bone conduction audiometry, however, has provided a quantitative measure of both the conductive and sensori-neural components of a deafness,[28, 29, 93] and the most sensitive test for the presence of conductive deafness is a demonstration of the absence of the occlusion phenomenon at lower frequencies.[95, 106, 118]

The discovery of the recruitment phenomenon[55, 117] and the recognition that its presence is indicative of end-organ lesions[44] facilitated the further subdivision of sensori-neural deafness into cochlear (i.e., end-organ) and retrocochlear deafness. There are various ways of measuring the recruitment phenomenon,[14, 55, 109, 121, 122] and it seems, on both theoretical[72] and observational grounds,[98] that the tests do not measure exactly the same thing. Thus, we may be led to recognize that certain cochlear deafnesses have particular audiometric patterns—for example, Martensson's series of dominant hereditary perceptive deafnesses,[98] where a positive recruitment phenomenon was indicated by impedence tests,[101] but where normal excursions were recorded on the Bekesy audiogram.[14]

Retrocochlear deafnesses can be further subdivided on the basis of the stability of the continuously recorded threshold as a function of time when a continuous tone stimulus is being employed. Perstimulatory threshold drift[129] appears to be a correlate of neuronal lesions.[76, 122]

Figure 2 shows schematically the determination of the locus of the shift in an auditory threshold. It should be noted, however, that *in practice, otoscopy, with the rest of the clinical examination, precedes audiometry.*

As was mentioned previously, audiometry affords a measure of the degree of hearing deficit. It is stressed, however, that this measurement applies to one particular aspect of a hearing deficit, i.e., a shift in the pure tone threshold of hearing. Although pure tone threshold shifts correlate with shifts in the speech reception threshold,[54] especially in the case of peripheral, and more so, conductive deafness, the more central the locus of the lesion, the poorer is the correlation. Moreover, speech discrimination may become impaired independently of shifts in thresholds of perception.[57] Consequently, speech audiometry[86] has a place in the quantitative measurement of hearing defects.

Not infrequently one wants to know whether a given hearing loss is organic, and in this respect both the Doerfler-Stewart test[45] and the delayed speech feedback test[47, 92] are helpful. More often, however, we wonder how much functional overlay there is in a particular case of deafness. A quantitative measurement of the "organic level" of the threshold of hearing can be obtained by EDR audio[21] (i.e., by psychogalvanic skin response) with respect to the peripheral pathway, and by EEG audio[41, 42] with respect to the entire auditory pathway, especially if the latter is used in conjunction with an electronic "averager."[131] Conditioning the electrodermal response (EDR) may, however, be difficult in aphasics.[60]

Objective tests for the presence of either conductive deafness or the recruitment phenomenon are also available. Impedance measurements on the ear,[101] using Lüscher's stapedius reflex,[96] may enable one to demonstrate the recruitment phenomenon.[75, 135, 136] Measurements from a probe

ANALYSIS OF LOCUS OF AIR CONDUCTION THRESHOLD SHIFTS

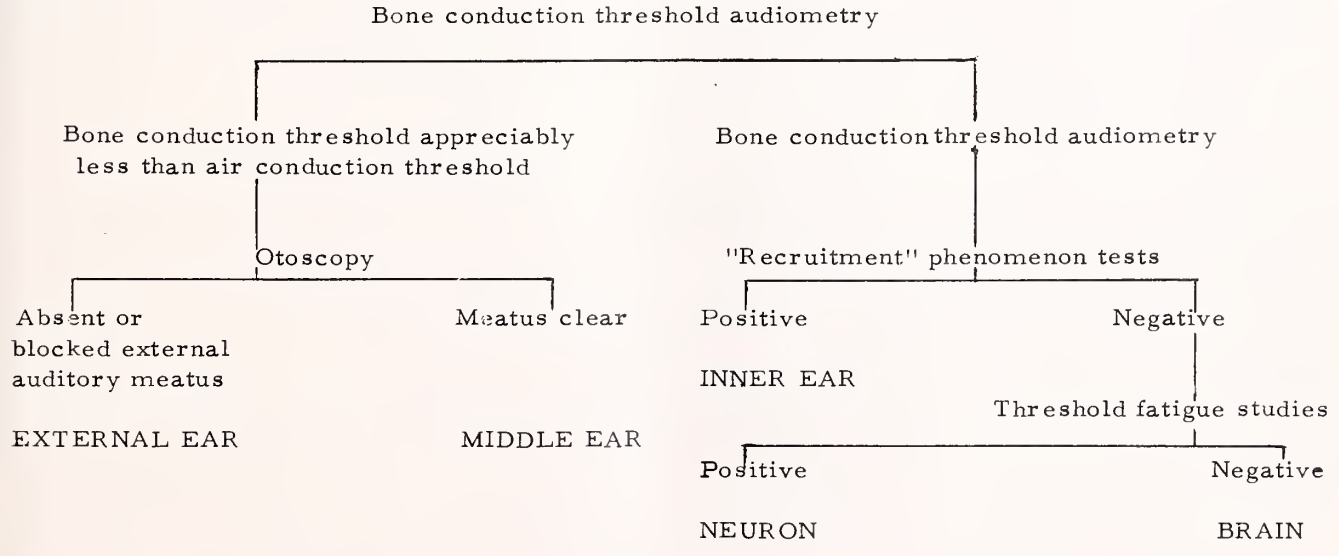

Figure 2.

tube microphone in the external auditory meatus, in conjunction with a transducer on the skull can indicate the presence or absence of a conductive deafness.[3]

(D) INVESTIGATION OF VESTIBULAR SYMPTOMS

Vertigo, as we shall define it, is the paramount and almost the sole symptom of dysfunction of the vestibular system. Vertigo is supposed to involve some hallucination of movement, on the part of the patient—the same phenomenon that laymen call "giddiness" or "dizziness." Thus *vertigo,* as it is used here, will cover rotatory sensations (the so-called "true vertigo"), sideways, back-and-forward and up-and-down sensations, unsteadiness in walking, and oscillopsia.[25] Symptoms of lightheadedness, faintness or fear of falling are not covered by this term. In our usage, the term *vertigo* will have the maximum diagnostic meaning and will be equatable with a deranged vestibular system.

Commonly, vertiginous patients complain of recurrent episodes of dizziness. A classification of the causes of recurrent vertigo is shown in Table 4. A single episode of vertigo that brings the patient to the doctor is usually a severe one, associated with nausea and vomiting. Such an attack can be termed "acute vestibular failure," in the sense that there has been a functional failure of the vestibular system. The causes of acute vestibular failure are shown in Table 5.

By far the commonest cause of recurrent vertigo, and indeed of vertigo in general, is Ménière's disease.[33, 100, 139] Notwithstanding many contrary statements, this condition is more commonly unilateral, and is commoner in people under the age of 45 years.[33] In its fullest expression, the condition is characterized by a unilateral, low-tone recruiting deafness, with a deranged caloric response. Many patients, however, have not sustained any cochlear or vestibular labyrinthine damage, so that the hearing and the caloric response are normal. In many cases, the vestibular dysfunction may be detectable only by electronystagmography.[8] Consequently, the diagnosis of Ménière's disease does not depend upon the demonstration of impaired hearing or abnormal vestibular responses to caloric or other tests.[39] The diagnosis depends primarily on the anamnesis.

The commonest cause of an acute vestibular failure is a condition that has been variously termed "vestibular neuritis,"[7, 20, 107] "vestibular neuronitis"[64] or "vestibular neuronopathy."[63] The condition is considered an inflammatory-toxic-allergic involvement of the vestibular neuron.[115] In one-third[43] to one-half[115] of the cases, there is radiological evidence of maxillary sinusitis. The only abnormality on neuro-otological examination is a derangement of vestibular responses that is compatible with a neuronal lesion, although a spontaneous nystagmus will be seen in the initial stages.[7]

Seven per cent of cases of vertigo seen at a neuro-otological clinic show a positional nystagmus.[33] This condition is produced by certain head positions.[11, 30] Two principal varieties are recognized. By far the commoner is the so-called benign paroxysmal positional nystagmus, and the other is referred to as positional nystagmus of the central type.

Benign paroxysmal positional nystagmus is a short-lived condition that is accompanied by ver-

TABLE 4

RECURRENT VERTIGO

End-Organ	Neuron	Brain Stem	Temporal Lobe	Cerebellum
Ménière's disease	Acoustic Neuroma	Basilar artery insufficiency	Epilepsy	Tumors
Circumscribed labyrinthitis[5, 114]	Vestibular neuropathy	Vertebral artery syndrome Due to:	Tumor	Abscess
		1) arteriosclerosis	Migraine	
Perilabyrinthitis[31]		2) cervical spondylosis		
Streptomycin intoxication		3) atlanto-occipital dysplasias	Episodic carotid artery syndrome	
Trauma		4) trauma	Encephalitis	
		Demyelinating disease		
Congenital syphilis			Anticonvulsants	
		Tumors		
		1) intramedullary-angioma	Functional	
Temporal bone tumors		2) extramedullary-meningioma; aneurysm		
		Syringobulbia		
		Anemia		
		Cerebral arteriosclerosis; Carotid sinus syndrome		

tigo, often severe, and it frequently includes the so-called rebound phenomenon when the patient is returned to the sitting position. As is implied by the term *benign,* the prognosis is good, although one case has been found associated with an astrocytoma of the cerebellum.[123] About half the cases of paroxysmal positional nystagmus are associated with aural disease.[43] The remainder are due to (a) vertebral artery compression,[84, 91, 116] and, or in association with, cerebral arteriosclerosis;[89] and (b) trauma.[30, 62, 119] The *modus operandi* is probably a sudden, unilateral partial vestibular failure.[94]

Positional nystagmus of the central type is due to lesions of the vestibular centers in the cerebellum.[30] It is characterized by nystagmus coming on as soon as the critical head position is reached, and continuing as long as that critical head position is maintained. This type of positional nystagmus is usually unaccompanied by vertigo in the critical head position. The associated lesion is usually a primary or secondary neoplasm, or multiple sclerosis.

The clinical examination of the vestibular system is based specifically on the examination of, and for, nystagmus, whether spontaneous or induced.[124] Considered physiologically, this implies an examination of the vestibulo-ocular reflex. Of the various reflexes in which the vestibular system participates (i.e., vestibulo-ocular, vestibulo-spinal and vestibulo-vegetative), examination of this reflex affords the most information on vestibular dysfunction. The presence of spontaneous nystagmus, with the exclusion of "endstellungs-nystagmus" (end-positional nystagmus) points to: (a) a defect in the eye or eyes involving an inability to acquire or maintain fixation (ocular nystagmus); (b) a lesion of the vestibular end-organ nerve or nucleus (vestibular nystagmus); or (c) a neurologic disorder involving either the mechanism of fixation or the central vestibular pathways (central nystagmus). The precise locus of the lesion responsible for a spontaneous nystagmus may be dependent upon an appraisal of the results of the neurological and neuro-otological examination.

The so-called caloric test[53] affords some quantitative measure of vestibular nerve function. The test may also indicate the presence of a phenomenon known as directional preponderance. This implies that nystagmus is more readily induced to one side than the other. Directional preponderance (DP) is regarded as a latent expression of spontaneous nystagmus.[82] The caloric test is made more sensitive by an electrical recording of the eye movements of the induced nystagmus, a procedure that is known as electronystagmography.[6, 65, 68, 87, 128] It is made possible by the eye's acting as a dipole,[38] where the cornea is positive with respect to the retina.[104] Consequently, a pair of electrodes placed on the face on either side of the eyes will register a difference in potential whenever there is a lateral eye movement. That difference will be proportional to the sine of the angle of rotation of the eyeball.[38, 52] In practice, however, a linear relationship is acceptable with gaze deflections of up to 30°.[130] The present employment of more sophisticated rotary-chair technics, using the Coriolis phenomenon, facilitates the testing of individual semicircular canals.[79] Eccentric fixation of the chair enables one to produce linear accelerations. By adjusting the direction of the linear acceleration, one can maximally stimulate either the utricular or the saccular otoliths.

(E) LOCALIZATION OF INTRACRANIAL LESIONS

Koranyi and Loeb's discovery of a directional preponderance of post-rotatory nystagmus in rabbits that had been subjected to unilateral hemispherectomy (1891)[88] heralded the application of neuro-otological tests to the localization of intracranial lesions. This observation was confirmed by Dusser de Barenne and de Kleyn,[12] who also showed that the same phenomenon occurred with caloric testing of the labyrinth. Subsequently, it was shown that the phenomenon occurred in hu-

TABLE 5

ACUTE VESTIBULAR FAILURE

End-Organ	Neuron	Brain Stem
Acute Pyogenic Labyrinthitis[5] (Otitic or Meningitic)	Vestibular Neuropathy (Vestibular Neuritis; Vestibular Neuronitis)	Posterior Inferior Cerebellar Artery Occlusion
Acute Serous Labyrinthitis	Cogan's Syndrome[35, 36]	
Acute Endolymphatic Hydrops		
Herpes Zoster Oticus[67, 73]		
Labyrinthine Hemorrhage		
Trauma		
Occlusion of Internal Auditory Artery or Branches		
Mumps Neuro-Labyrinthitis		

man beings with brain tumors,[13, 85] especially with tumors of the posterior part of the temporal lobe.[53]

Recently, it has been shown that the intensity of the nystagmic response to caloric stimulation as a function of time shows different patterns,[137] dependent on the locus of a vestibular nerve lesion.[116] The normal pattern, which gradually rises to a maximum and then falls off, has been termed the "culmination phenomenon."[137]

A phenomenon analogous to auditory recruitment may occur with lesions of the vestibular labyrinth. This has been termed vestibular recruitment, and it may occur with either caloric[9, 10, 58] or rotatory stimuli.[1]

Bárány introduced examination of optokinetic nystagmus into neuro-otology more than 50 years ago. Optokinetic nystagmus (i.e., railway nystagmus) is an ocular and not a vestibular nystagmus, but as Cawthorne *et al.* say,[32] the fact that certain of its characteristics had long claimed for it an intimate relationship to the vestibular system offered a good reason for its inclusion in the diagnostic armamentarium of the neuro-otologist. Fox and Holmes[56] showed that directional preponderance of optokinetic nystagmus occurs in a large proportion of lesions of the inferior part of the parietal lobe. As with caloric induced nystagmus, optokinetic nystagmus can be recorded electronystagmographically.[46] This technic provides an objective, quantitative and more sensitive measure of the response.

Patients with unilateral temporal-lobe lesions may show essentially normal pure tone and speech audiograms. However, some six years ago, Bocca and his associates showed that the intelligibility of speech passed through a low-pass filter, and presented monaurally, was impaired in contralateral temporal lobe tumors.[18, 19] Other types of distorted speech are also less intelligible when presented to the ear opposite to a temporal lobe with a functional deficit.[17, 26] In addition to this impairment of contralateral distorted-speech intelligibility in temporal lobe tumors, a specific loss of learning ability by auditory means is said to occur when the dominant hemisphere is involved.[102] Jerger[77] has shown abnormalities of median plane sound localization in subcortical lesions, and Matzker[99] has shown that dichotic speech integration is impaired in brain stem lesions.

Particular neuro-otological patterns of localizing value are shown in Table 6.

In addition to these neuro-otological abnormalities in organic disease of the nervous system, different reactions occur, or may occur, in mental disorders. Vestibular reactivity is diminished in schizophrenics[34] and enhanced in neurotics. Neurotics also show evidence of a different behavior to the cross-masking effect of pure tones.[74]

TABLE 6

NEURO-OTOLOGIC "SYNDROMES"

Locus	Pathway	
	Auditory	Vestibular
End-Organ	Recruitment phenomenon	(1) Canal paresis; culmination phenomenon may be present (2) Vestibular Recruitment
Neuron	Per-stimulatory threshold drift	Absent culmination phenomenon
Nuclei	?	Plateau type culmination phenomenon
Cerebellum	No abnormality	Central type positional nystagmus
Brain Stem	Impairment of dichotic speech integration	Deranged optokinetic nystagmus
Temporal Lobe	(1) Impairment of contralateral distorted speech intelligibility (2) Specific loss of learning ability by auditory means when dominant hemisphere involved	Ipsilateral directional preponderance of caloric induced nystagmus
Parietal Lobe	No abnormality	Ipsilateral directional preponderance of optokinetic nystagmus

(F) AS A COMPLEMENT TO THE GENERAL NEUROLOGICAL EXAMINATION

All except four (II, III, IV and VI) of the cranial nerves can be claimed to be in the ENT area, and as a corollary to this claim it can be asserted that the neuro-otologist has developed or is using quantitative methods for measuring certain cranial-nerve functions. These are shown in Table 7. Moreover, with respect to some cranial nerves, for example the vagus, it may be possible only for someone skilled in performing an otolaryngological examination to make an accurate assessment of a nerve's functional integrity. A neuro-otological examination may, therefore, complement a general neurological examination.

With respect to the clinical examination of these cranial nerves, one or two points are of note. Loss of the corneal reflex is often the first manifestation of fifth-nerve involvement. False positive diagnoses of facial palsy are frequently made because of the prevalence of slight degrees of facial asymmetry. This shows as a nasolabial groove that is more prominent on one side than on the other.

TABLE 7

QUANTITATIVE INVESTIGATION OF CRANIAL NERVES IN ENT AREA

Nerve		Procedure
I		Olfactometry[23, 80]
V		Quantitative Corneal Sensibilometry[16]
VII	(a)	SPECIAL VISCERAL EFFERENT NEURONS
		Electromyography[111, 133] and Intensity-Duration Curves[22]
	(b)	GENERAL VISCERAL EFFERENT PATHWAYS
		(i) CHORDA TYMPANI
		Submandibular salivary flow[97]
		(ii) GREATER SUPERFICIAL PETROSAL
		Quantitative measurement of Naso-Lacrimal Reflex[140]
	(c)	SPECIAL VISCERAL AFFERENT PATHWAYS
		Clinical quantitative gustometry[69]
		Electrogustometry[90]
VIII	(a)	COCHLEAR
		Audiometry[72, 93]
	(b)	VESTIBULAR
		Caloric electronystagmography[6, 65, 68, 87]
IX		SPECIAL VISCERAL AFFERENT PATHWAY
		Clinical quantitative gustometry[69]
		Electrogustometry[90]
X		High Speed Cinematographic Studies of Larynx[51, 126]
		Electromyography of Intrinsic Laryngeal Muscles[24, 49]

One of the earliest signs of unilateral facial palsy is apparent when one notices that the eyelids are not buried so much on the involved side as on the normal side when the patient has been asked to screw up his eyes. Wasting in the region of the sternoclavicular attachment of sternocleidomastoideus is a valuable diagnostic sign with respect to the spinal accessory nerve.[110] With respect to the hypoglossal nerve, the essential sign of a lower motor-neuron lesion is wasting of the tongue, and if this is absent, any apparent fibrillation or deviation of the tongue must be ignored.[110]

(G) DISCOVERY OF LESIONS THAT ARE PRIMARY TO THE NEUROLOGIC DISORDER

Characterizing this group, we have otorhinogenous brain abscesses. About half of the brain abscesses are due to paranasal-sinus or mastoid infection. They are characterized by the triad of headache, vomiting and stupor.[112] Headache can be the earliest and most constant feature of all otogenous intracranial complications.[113] Even the fact that the patient himself is unwell may mean that suppuration has extended beyond the middle-ear cleft.[40] Papilledema is uncommon in acute brain abscess.[112] Pennybacker's series of rhinogenous frontal-lobe abscesses[112] were all associated with acute sinusitis and signs indicative of frontal osteitis. Epileptic episodes occurred early. Otogenous temporal-lobe abscesses were characterized by pain and usually a visual-field defect on perimetry. Cerebellar abscesses were characterized by a central type of spontaneous nystagmus, and the head was bent forwards, in contrast to the opisthotonoid attitude of cerebellar tumors. In 95 per cent of cases, the cerebrospinal fluid shows an increase in protein and/or cell content. Cerebral abscesses do not differ essentially from neoplasms in their electrical behavior, but delta waves tend to be exceptionally slow, irregular and of high voltage. They are also more widespread, probably because of the extensive inflammatory edema.[120]

(H) EXTRANEURAL EXTENSION OF TUMORS INVOLVING THE NERVOUS SYSTEM

As one example of this category, we can cite the presentation of chordomata in the postnasal space.[66]

VALIDITY AND SENSITIVITY OF NEURO-OTOLOGIC TESTS

As is the case with the majority of diagnostic tests in general, the validity and sensitivity of many neuro-otologic tests have yet to be determined. This applies particularly to the phenomenon of directional preponderance (DP). In respect to caloric DP, it has been claimed that the prevalence of DP in Ménière's disease is the same as that in the general population.[81] Moreover, from a study of intracranial disorders, correlating caloric DP with EEG and other special investigative results, Kirstein and Preber[83] claimed that the caloric DP had no unequivocal localizing value. In a study of 130 patients with supratentorial lesions demonstrated by operation, ventriculography or arteriography, Andersen[2] showed that temporal-lobe involvement was not invariably associated with a caloric DP, and that a DP may occur in the absence of involvement of the temporal lobe. In respect to optokinetic DP, lesions of the parietal lobe may occur unaccompanied by defective optokinetic nystagmus.[37, 108, 138] In fact, Roelofs[125] states that the angular gyrus has nothing to do with the optokinetic pathway. All phenomena which seem to contradict this assertion appear because of lesions or irritations of this pathway in the surrounding regions because the cortifugal fibres collect under the gyrus angularis before proceeding to the internal capsule.[125] Using electronystagmography in a recent study of 58 brain lesions, Enoksson[46] showed that optokinetic nystagmus was disturbed by pathologic processes of widely varying localization. However, as with the caloric DP in other studies, optokinetic DP did appear to be of lateralizing, if not of localizing, value.

Neuro-otological localizing tests also have yet to be satisfactorily compared with neurological and neuro-ophthalmological localizing tests. There was evidence that a caloric DP might not be so sensitive an index of posterior temporal-lobe tumors as are visual-field studies,[4] but a subsequent study

of a larger series did not confirm that possibility.[127]

It has recently been shown that tests of auditory and visual recent memory are not valid measures of temporal-lobe function, but that impairment of those processes is dependent upon damage to the subjacent hippocampal complex.[132]

It may also be asked, in these days of electroencephalography, whether neuro-otologic tests for localizing brain lesions are redundant. However, there is evidence that EEG appraisals may themselves be open to a certain amount of inter-observer variation.[15]

Clearly, further studies are yet required to assess the validity, accuracy and sensitivity of neuro-otologic tests, especially with regard to those aimed at localizing brain lesions.

NEURO-OTOLOGY IN RELATION TO OTHER SPECIALTIES

The scope of neuro-otology, as indicated here, is not too restricted to constitute a separate specialty. Indeed, the field has grown so rapidly that, in many countries, some of its subdivisions—e.g., "audiology" and "speech pathology"—are themselves recognized as distinct specialties. In recent years, inroads have been made into otolaryngology by allergists, radiotherapists, endoscopists, plastic surgeons and oncologists. There are conscious or unconscious fears among otolaryngologists that the recognition of neuro-otology as a separate specialty would produce a situation in which, if the orthopedist were then to claim mastoid and stapes operations as facets of bone and joint surgery, otolaryngology would disappear as a separate entity. Such, however, need not be the case. Neuro-otology should stand in the same relationship to otology as neurology does to neurosurgery. The emergence of neuro-otology should, therefore, strengthen rather than weaken the otolaryngologic specialty.

SUMMARY

The functions and scope of neuro-otology have been described, and its relationship to the otolaryngologic specialty has been discussed.

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Gastritis

A Symposium Presented to the Scott County Medical Society

Dr. Merle J. Brown: In selecting a topic for symposial coverage at this meeting, the Program Committee was swayed by four considerations: (1) Gastritis is being discussed frequently at medical meetings and in the literature. (2) There are some controversial aspects of the matter. (3) The symptomatology may be difficult for the clinician to interpret. (4) A general review of the subject seems apropos at this time.

The participants in tonight's discussion are Dr. Robert A. Towle, of Davenport, a gastroscopist who will act as moderator; Dr. Newell T. Braatelein, a member of the Quad Cities Pathologists group stationed at Moline Public Hospital; and three men from the faculty of the S.U.I. College of Medicine, Dr. John T. Keller, of the Department of Radiology, Dr. James A. Clifton, of the Department of Internal Medicine, and Dr. Robert T. Tidrick, of the Department of Surgery.

Dr. Towle: I am sure that the classifications of cases and the nomenclature that we should employ are some of the things that should be made clear to us. For these ground rules, we should call upon the pathologist.

TABLE I
TYPES OF ACUTE GASTRITIS

I. Simple
A. Alcohol
B. Drugs—salicylates, iodides
C. Tea, coffee, highly-seasoned foods
II. Corrosive
A. Phenol and mercuric chloride—coagulation
B. Sodium and potassium hydroxide—softening
C. Acids—charring and perforation
III. Infectious
A. Phlegmonous or purulent—streptococcus or coliform
B. Necrotizing
C. Hemorrhagic—complicating diphtheria or pneumonia

Dr. Braatelein: I want briefly to review the various types of gastritis. In the acute category, the subclasses are the simple, the corrosive and the infectious (Table 1).

One can say that on any given day in this country, the commonest form of acute gastritis is the alcoholic. In addition to the so-called "simple gastritis" due to alcohol, however, we have certain drug-induced types resulting from salicylates or iodides, and some for which coffee, tea or highly-spiced foods have been responsible.

The pathology of this type of gastritis is not well known. I am sure that very few of my listeners were confronted on New Year's Day by the alcoholic type of gastritis, and indeed the only time when we pathologists are likely to see such a case is when an individual has died after some acute alcoholic episode, or when we chance to do an autopsy upon someone who has been overly fond of coffee, tea or highly-seasoned food. The pathology that has been described consists of hyperemia with very minimal inflammatory changes.

The second subtype of acute gastritis is, of course, a very serious one. There are three types of changes, generally speaking, that are involved in corrosive gastritis. Phenol and mercuric chloride tend to cause coagulation of the gastric mucosa; sodium and potassium hydroxide tend to cause softening and consequent sloughing of the mucosa; and acids—particularly the strong acids such as sulphuric—will cause charring, with complete necrosis of the wall and subsequent perforation. Now if a patient who has ingested one of these substances chances to live, the end result is scarring, possibly to such an extent that surgery is indicated to relieve an obstruction. The acute pathology is of interest only from the autopsy standpoint. In

the acute phase, there is very little that can be done for the patient other than symptomatic treatment.

Infectious gastritis is perhaps of more interest to general practitioners. Of the conditions of this sort that are recognizable grossly, the phlegmonous type is usually due to a streptococcic infection associated with another infection elsewhere in the body. It is a condition in which the entire stomach is usually involved. The mucosa is flattened; there may or may not be a small amount of exudate on the surface; there may or may not be ulceration of the mucosa. But the main pathology is located in the submucosa, which is tremendously thickened, and there are focal areas of inflammatory reaction, with possible abscess formation. Associated with this is thrombosis of the vessels, and actual microabscesses may have formed in the walls of the stomach. In this condition, one can usually culture either a streptococcus or one of the coliform organisms.

The next classification is a little more nebulous. Necrotizing acute gastritis has been described in association with Vincent's angina, when the organism has been said to be a spirochete or a fusiform bacillus. Patients suffering this ailment tend to develop rather sharply punched-out ulcerations which frequently perforate, and in the few cases that have been described, the prognosis has been shown to be very poor. The necrosis is nonspecific, and there is little that can be done.

The last type is one which we see rarely. I include it more or less because it has been mentioned frequently. It is known as acute hemorrhagic gastritis, and particularly in the older literature it was listed as a complication of diphtheria or pneumonia. It is associated with multiple mucosal hemorrhages, and apparently in some cases gastric hemorrhage did result from this type of process.

Next, let us discuss chronic gastritis (Table 2). The literature is a little bit vague on this phase of the subject. Some people divide it into chronic gastritis and chronic atrophic gastritis, but from a morphologic standpoint I have found nothing to make me believe that there are two distinct entities. In the first place, atrophic gastritis is very frequently an accompaniment of such diseases as pernicious anemia and carcinoma of the stomach. An interesting feature is that the gastritis in these cases tends to be limited to the cardia and fundal portion of the stomach.

TABLE 2
TYPES OF CHRONIC GASTRITIS

I. Atrophic—associated with specific diseases, e.g., pernicious anemia and carcinoma
II. Hypertrophic
III. Granulomatous
A. Syphilitic
B. Tuberculous
C. Eosinophilic

A separate type of atrophic gastritis that is associated with the process of aging is most frequently seen in the pyloric end of the stomach. It is one that, over the past few years, has occasioned a great deal of interest in all of the people who are working with this disease because it frequently is a precursor of carcinoma.

Pathologically, atrophic gastritis shows flattening of the mucosa, but the mucosa is usually intact. The gastroscopist describes prominence of the mucosal vessels, a characteristic which the pathologist, of course, seldom can see by the time he gets the specimen, and although the mucosa may be of normal thickness, it usually is somewhat thin and, microscopically, it shows infiltration of chronic inflammatory cells with a rather remarkable change in the mucosa consisting of a metaplasia of the mucosal glands to those of an intestinal type, and disappearance of the parietal cells and chief cells. Naturally, this is associated with a reduction in the amount of free hydrochloric acid in the gastric secretions.

Hypertrophic gastritis is a very interesting condition in that there appears to be some disagreement about just what constitutes it. There appear to be two types, both of which show tremendous thickening and increased prominence of the gastric rugae. On biopsy, most of these cases show what appears to be relatively normal mucosa. There are some instances, however, in which there is some evidence of glandular hyperplasia, with an associated inflammatory reaction. The thing that poses difficulties for the pathologist, in his attempts at evaluation, is that *ordinarily* the gastric mucosa contains some inflammatory cells, and thus it frequently is difficult to be certain whether any significant degree of inflammatory reaction has occurred.

The last classification, granulomatous gastritis, is associated with syphilis and tuberculosis. Syphilis, of course, can cause a condition resembling linitis plastica, and once in a while a rather remarkable case comes along. The surgeon resects the stomach for a diagnosis of carcinoma, and on examination finds the lesion to be an inflammatory ulceration, with thickening and fibrosis of the stomach wall. The microscopic appearance isn't specific for syphilis, but is that of an inflammatory process. The serology, as a rule, will solve the problem. Tuberculosis can occur in three forms, and the one that is most difficult is the one that involves the stomach rather diffusely. The microscopic appearance is that of classical tuberculosis, and there almost always is evidence of tuberculosis elsewhere.

The eosinophilic granuloma is a rather unusual condition that has occasioned a great deal of interest because of the fact that it appears to be some type of unusual hyperplasia of the mucosal con-

nective tissue associated with large numbers of eosinophils that tend to form a polypoid mass, and at least in some cases the mass has been known to herniate into the lumen, and to project through the pylorus, causing partial or complete obstruction.

One type of gastritis is not listed in Table 2. In addition to the gastritis that accompanies carcinoma and ulceration, there is one that follows gastric resection, gastroenterostomy and other operations on the bowel. I must thank Dr. Towle for calling my attention to it.

Dr. Towle: I rather insisted upon the inclusion of "gastritis with other conditions" because of some points that are to be made later on, particularly concerning gastritis in conjunction with tumor, ulcer or postoperative stomach. Some people have placed post-irradiation gastritis in this classification, though cases of that sort very rarely come to the clinic or to the gastroscopist.

The acute gastritides are very seldom seen in general practice or in the practice of internal medicine. Of the last 500 gastroscopies that I have performed in Davenport, there have been just three on patients who had been referred to me by such physicians. The patients are all younger people, their histories have been short (from 72 hours to one week), and they all are sick. Occasionally this acute gastritis has involved the entire wall of the stomach, and sometimes it gets the patient to the surgeon. Such individuals present to the gastroscopist an identical pattern of redness, swelling, denudation or superficial ulceration, and they have inflammatory membranes that are ragged, yellow and white, and glistening.

These people don't have the so-called "dyspeptic histories." They are young people. They are in pain. Their abdomens are often acute. Of the three whom I have seen most recently, the last one was very ill indeed. This is not the surgical emergency of the acute abdomen, which usually presents with vomiting of purulent material.

I want to comment briefly on the two most important of the chronic gastritides, both of which are very easy to spot when we are dealing with the physiological-pathologic conditions prior to the disruption of blood supply. When one gastroscopes chronic atrophic gastritis in the stomach of the pernicious anemic patient, he sees blood vessels that never show in a normal stomach. They are submucosal vessels, and they are visible mainly because of loss of chief cells from the mucosa. The stomach doesn't show free acid, even after histamine. There is a greenish cast. The mucosa is ironed out. The rugae are raw.

One notices some small polyps, rather than cobblestoning, and they are true adenomatous polyps in an area of chronic atrophic gastritis. This we shall come back to later, for the internist believes that chronic atrophic gastritis is the most important of the gastritides, and is definitely a lesion in the stomach that must be watched as a possibly precancerous lesion. Statistics have shown that there are more carcinomas in pernicious anemia stomachs, and certainly about four times more adenocarcinomas of the stomach occur in atrophic gastritis patients than in the general run of hospital admissions.

IS "GASTRITIS" A WASTEBASKET DIAGNOSIS?

Dr. Clifton: Gastritis is a very difficult subject for me to talk about. I am not an enthusiastic supporter of this disease entity; rather, I am inclined to pour cold water upon it. I don't believe that a great many symptoms are due to this condition.

I am glad that Dr. Towle talked about acute gastritis, for I don't intend to say very much about it, except to comment that I don't often see cases of it either.

There is a great deal of controversy in the literature—and confusion in my mind, and probably in yours—concerning gastritis. Though clearly they are wrong, there are many competent observers who believe that there is no such thing. On the contrary, Dr. Braatelein's classification is a very fine one. The very complicated classifications which many people, mainly Schindler, have put forth are responsible for one of the problems in connection with this disease. These types which Dr. Braatelein has outlined do exist, but one question is whether these conditions produce symptoms, and a question of even more importance is whether they produce a definite clinical syndrome. I don't believe we know the answers entirely.

It seems to me that the weight of evidence at present tends to show that chronic gastritis seldom causes symptoms and probably doesn't have a definite and characteristic symptom complex. I think that we clinicians have wronged gastritis by making it a wastebasket diagnosis. We have diagnosed it on many occasions without a shred of evidence that gastritis was indeed present. Few of us do this sort of thing in evaluating patients with diabetes or myocardial infarction, but we fall into the trap of making this diagnosis with patients who come to us with symptoms of dyspepsia, fullness after meals, heartburn, vague upper-abdominal distress, nausea, flatulence, etc. The radiologist tells us that the upper gastrointestinal series is negative. Then we have the patient gastroscoped, and the gastroscopist reports that there are a few scattered petechial hemorrhages in the stomach, that there is some adherent mucus and that there is a little redness. This, in our minds, substantiates the diagnosis of gastritis. We so inform the patient, and we prescribe therapy. Unfortunately, I think we are often in error in every step of this diagnostic triad. That is to say, we are in error if the final authority is the histology. We have to accept a final authority, and it seems proper to me that it should be the histology.

As far as I can ascertain, the foundation for this structure of error was laid in the 1930's. The early gastroscopists described many different types of

mucosal changes and labeled them as separate types of gastritis. To each of them they attached a cluster of symptoms which they said were specific for that particular type. In the enthusiasm, and even the awe, which surrounded the introduction of the gastroscope, clinicians failed to look critically at this early work. Some pathologists voiced words of caution, but I believe they were unsuccessful simply because they did not have available to them enough specimens of fresh gastric mucosa to permit their studying gastritis in its broadest aspects. We all are aware of the rapid autolysis that takes place in the gastrointestinal mucosa following deprivation of its blood supply, and therefore specimens examined at autopsy and after surgery do not represent, in most instances, the true state of the mucosa.

Finally, some people began to question the relationship of gastritis to symptoms. I think that Dr. Walter Palmer and his group in Chicago were among the first to call our attention to the fact that there was a dearth of correlation between symptomatology and what was being seen through the gastroscope. That group published a number of papers on the subject in the 1940's, clearly showing that in their hands there was *not* a good correlation between the gastroscopic findings and the symptomatology.

In the 1950's, Doig and Wood, in Australia, devised an instrument for taking biopsies from the mucosa in the stomach of the living, unanesthetized patient. That, in my opinion, has been the most significant forward step in helping to clarify this still very confusing subject. Those investigators published numerous papers on this topic, and their work was summarized in a very nice article in a 1955 issue of the QUARTERLY JOURNAL OF MEDICINE, presenting their results from the examination of some 1,000 biopsy specimens. They extended the observations of Dr. Palmer's group by correlating symptomatology, radiology, gastroscopy and pathology.

The first thing they did was to take away from us—i.e., the gastroscopists—the diagnosis of hypertrophic gastritis. We had been accustomed to seeing cobblestoned mucosa that appeared to have large rugae and a few scattered petechial hemorrhages, and diagnosing hypertrophic gastritis on the basis of that evidence. In some clinics, hypertrophic gastritis had made up 40 per cent of the gastroscopic diagnoses. Obviously, the pathologists never saw hypertrophic gastritis in such large quantities, and they had already begun to question the correctness of the diagnosis. Doig and Wood showed conclusively—and it has similarly been shown by others including Eddy Palmer—that the vast majority of people with a gastroscopic appearance of hypertrophic gastritis have normal mucosa on gastric biopsy. In fact, they were unable to find *any* cases of hypertrophic gastritis in their series of patients. It seems to me that this diagnosis has been eliminated from our consideration, except in the patients who have true giant hypertrophic gastritis—an extremely rare condition.

Doig and Wood felt that there is a correlation—albeit a slight one—between pathology and symptomatology, particularly in those patients who have severe superficial gastritis or atrophic gastritis. Other investigators have come to the conclusion that there is no real correlation between pathology and symptomatology. In fact Eddy Palmer concluded that the symptomatic form of gastritis isn't gastritis at all, but is a functional disease resulting from what he called "tense stomach." Now I hasten to say that although gastritis can be eliminated as a cause of symptomatology in these people, I don't think we have any proof that the true cause is a tense stomach. We may infer that this so-called functional dyspepsia is due to a neuromuscular imbalance in the stomach, but as yet there is no proof of it. At any rate, it doesn't appear that the clinician can diagnose gastritis from the history with any degree of accuracy.

Furthermore, in many cases, the diagnosis can't be reliably made from gastroscopy. Doig and Wood found that gastric mucosa which appeared abnormal when viewed through the gastroscope was, in many instances, normal histologically. Usually, the gastroscopist can correctly diagnose true gastric atrophy and severe superficial gastritis. As I have already pointed out, the gastroscopist's "hypertrophic gastritis" has no histological counterpart. I think that gastroscopists have overdiagnosed gastritis because they have not appreciated the wide range of appearances that the normal gastric mucosa can take. On the other hand, they have underdiagnosed the cases where the mucosa looks normal and is histologically abnormal, simply because gastroscopy has certain very definite limitations. It isn't a good enough technic.

What about radiology? I don't think that the radiologist can make a diagnosis of gastritis. I have seen many patients, as all of you have, in whom the diagnosis of antral gastritis has been made by the radiologist. As far as I can tell, this has usually been on the basis of irritability in that area, a widening of the mucosal folds, and a failure to achieve what is described as a normal mucosal pattern. Where studies have been done on this point, with gastroscopy and biopsy, in no cases have there been any substantiations of the radiologic diagnosis. Now it is clear that some type of disturbance is present. I think it is also clear that the difficulty has been a deformity of the x-ray resulting from neuromuscular mechanisms. It is not the result of inflammation of the mucosa of the stomach. In a long-forgotten but excellent paper, a Swedish radiologist named Forsell, in 1923, showed that the folds of the gastric mucosa are dependent upon the action of the muscularis mucosa for their size and configuration. This changes from moment to moment, from hour to hour, and from day to day. It is not dependent, in the vast ma-

jority of cases, upon changes in the mucosa itself.

Since I have eliminated the radiologist, spurned the clinician and cast doubt on the gastroscopist, how can we make an accurate diagnosis of gastritis? It seems to me that, as is so frequently the case in medicine, we come to our final judge and arbiter, the pathologist. Through the use of one of the many modifications of the Doig and Wood instrument that are available, we can secure biopsies of the gastric mucosa and establish the diagnosis pathologically. At S.U.I., we have used the Rubin modification and found it very satisfactory. The procedure takes only a few minutes and carries little risk. Hemorrhage has been reported in patients with gastric atrophy, but it is extremely rare.

As far as therapy is concerned, I don't believe that we need to say very much. Gastric atrophy, as such, requires no therapy. Superficial gastritis and atrophic gastritis are conditions which seemingly respond to the usual symptomatic type of treatment that we give them. The results of therapy are difficult to judge, since the gastritides tend to come and go, as does peptic ulcer.

X-RAY DIAGNOSIS HELPS IN SOME INSTANCES

Dr. Keller: The diagnosis of gastritis presents many problems to the radiologist because of the many types and degrees of involvement. The gastroscopic examination is more reliable, but in many cases a roentgenographic exam can be of great help.

Now for the radiographic appearance of the various types of acute gastritis. The simple superficial type, we don't feel can be well demonstrated by radiology. There may be an unusual amount of mucus present, but this factor is too indeterminate to be of much practical value. As for the corrosive type, in the few cases that we have seen, GI series done shortly following the swallowing of corrosive liquid showed an ulcerated, frayed, ragged appearance of the stomach wall. Later on, after the acute phase had subsided, many of the cases go on to narrowing and even obstruction of the distal stomach, and simulate the rigid, unyielding appearance of scirrhous carcinoma.

Figure 1 shows the stomach of a 62-year-old man who had swallowed 20 per cent hydrochloric acid. As you can see, almost the entire midportion and distal portion of the stomach have been extremely narrowed and scarred down. The film was taken about four weeks after the swallowing.

Figure 2 shows a 76-year-old man who had scirrhous carcinoma of the stomach. There is the appearance of extreme scarring and infiltration of the distal two-thirds of the stomach. The duodenal bulb seems to be within normal limits. I have shown these two examples to demonstrate that radiographically we can't tell the difference between the results of corrosives, after they have had time to scar down, and scirrhous carcinoma.

In the phlegmonous type of infectious gastritis, the mucosa is intact over the involved area, but there is a swelling of the mucosal folds. The flexi-

Figure 1. Stomach four weeks after ingestion of 20% hydrochloric acid.

Figure 2. Scirrhous carcinoma of the stomach.

bility of the stomach wall is impaired, and there will probably be gastric retention. The involved portion of the stomach may show localized narrowing, and some cases show an annular smooth filling defect of the pylorus. In some slender patients, it may be possible to demonstrate the thickening of the gastric wall. It shows up as a soft-tissue shadow next to the ingested barium meal.

In chronic gastritis, the atrophic type will show rugae that are flattened and thin, and they may even be obliterated. The mucosal folds may be difficult to visualize at all. This is not a very reliable roentgenologic diagnosis, since many normal stomachs will show a similar appearance. In hypertrophic gastritis, the mucosal rugae are fewer in number. They are rigid, tortuous and enlarged—broader than usual and elevated. Granular, nodular and warty formations may be seen, and as these get bigger they may produce a pseudopolyposis which shows as filling defects in the stomach. Sometimes the pylorus becomes narrow and rigid, and the cardia may show malignant-appearing defects due to hypertrophied folds.

Figure 3 shows the stomach of a 56-year-old man who had had severe burning epigastric pain, precipitated by eating, for a period of five months. The GI series showed these large gastric rugae. The stomach was supple to palpation, however, without the rigidity usually associated with carcinoma. We felt that hypertrophic gastritis was the most likely diagnosis, but could not rule out a lymphoma or a carcinoma. Surgical inspection showed that it was probably a lymphoma, so the patient was closed up without a resection. The final sections, however, showed giant hypertrophic gastritis.

Figure 4 shows a patient with lymphoma of the stomach, metastatic from lymphosarcoma of the tonsil, and one can see that on the greater curvature there are large defects simulating the large rugae that we saw in Figure 3.

Figure 5 was thought to represent a lymphoma on the basis of an upper GI series. The antral region shows great enlargement of the rugal patterns, and it was considered very rigid to palpation. At operation, however, it proved to be hypertrophic gastritis, rather than a lymphoma.

The granulomatous lesions such as gastritic syphilis are not pathognomonic, but do furnish evidence of gastric disease. Three types of gastric syphilis are generally recognized. There is the diffuse infiltrating type which produces a small, rigid, linitis plastica type of stomach, which empties quickly. Figure 6 shows the stomach of a 60-year-old female who had had complaints of epigastric pain for two years. It grew constantly worse, and she also had sub-sternal pain several hours after eating. She had little or no nausea, but she would vomit undigested food one hour after eating. The GI series showed a rigid, narrow pars media of the stomach. The bulb was within normal limits. The impression was scirrhous carcinoma of the stomach, and a subtotal resection was done. The pathologist's report showed syphilitic gastritis. The serology was positive, and the patient was treated for lues.

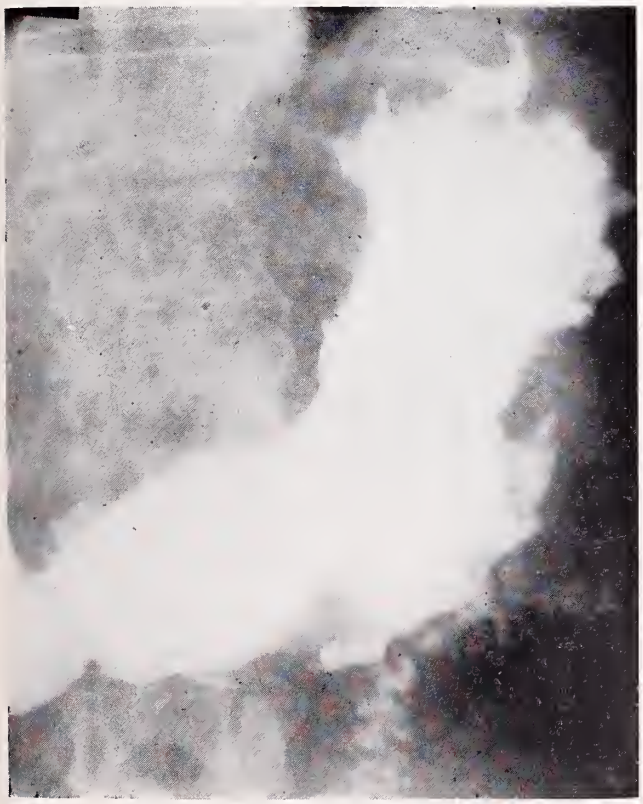

Figure 3. Giant hypertrophic gastritis.

Figure 4. Malignant lymphoma metastatic to the stomach.

The second type of syphilitic gastritis is an ulcerative type that looks like the usual peptic ulcer, but instead of an incisura, it shows an hourglass type of deformity. The third type of syphilitic gastritis is the gummatous type which presents a filling defect, usually involving the pyloric region and simulating a carcinoma.

There is one area where I believe the radiologist has a better chance than the gastroscopist of diagnosing gastritis, and that is in the antrum. Antral gastritis, according to our radiologic texts, is characterized by hypersecretion, pylorospasm, antral spasm and narrowing of the antrum. The gastric folds are enlarged, and have a polypoid appearance. There sometimes is prolapse of the gastric mucosa into the duodenum, and delayed emptying time. Differentiation from other lesions, especially carcinoma, is difficult. The most important factor is a careful study of the rugae. One should be able to trace them through the involved area without interruption, and without any change in their architecture. This is best done by spot films with different degrees of compression.

In general, then, we feel that the roentgenographic exam is of little help in the diagnosis of acute superficial and chronic atrophic gastritis. It can be useful in hypertrophic chronic gastritis, but it is far from infallible. It can be of much assistance in antral gastritis, since that area may often be inaccessible to the gastroscope.

As regards x-ray therapy, the only useful applications that occur to me would be a gastritis associated with a peptic ulcer with hyperchlorhydria. One might give x-ray therapy to the stomach with the intention of reducing the secretion of hydrochloric acid, thus helping the gastritis indirectly.

Figure 5. Hypertrophic gastritis simulating lymphoma.

SURGERY IN CASES OF GASTRITIS

Dr. Tidrick: In reviewing the indications for surgery in cases of gastritis, let's go back to Dr. Braatelein's classifications. They are both practical and simple.

Unless there are some unusual complications such as hemorrhage—and that isn't a common manifestation—no surgical treatment needs be used for plain and simple acute gastritis. Next, as regards the corrosive. All of our speakers have referred to corrosive gastritis and its complications, in one way or another. If it is perforated, or if there is massive bleeding, or in the late stage if there are scars and obstruction, surgical therapy may be necessary. Next, the infectious type. I have had no experience at all with phlegmonous gastritis. At a meeting in Cincinnati a couple of years ago, one of our surgical colleagues presented three cases of phlegmonous gastritis which had been treated surgically. At least two of the patients had survived, but this is ordinarily a highly fatal condition. So there may be some place for surgery in the treatment of this subtype of the disease, but I can't speak from first-hand experience. As for necrosing gastritis, again I have had no personal

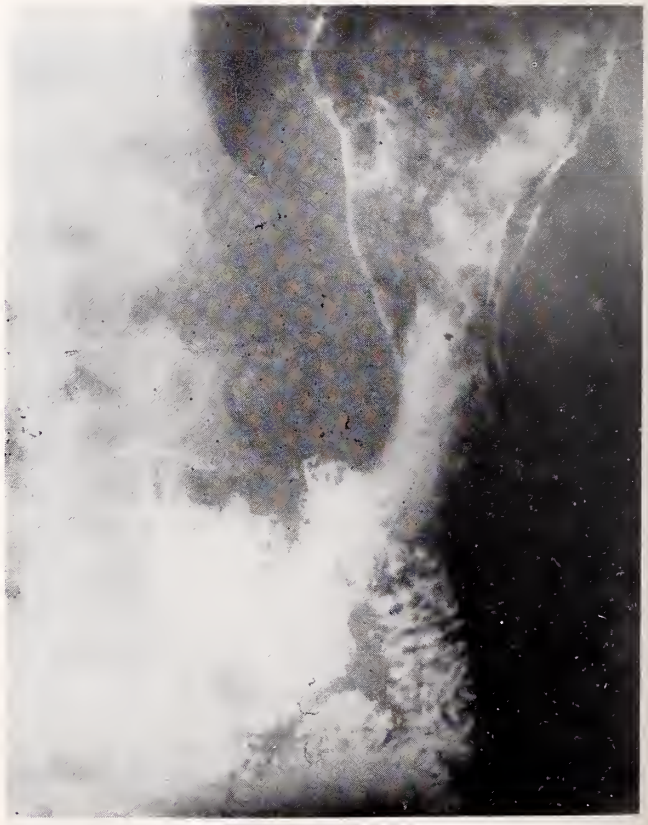

Figure 6. Syphilitic gastritis simulating scirrhous carcinoma of stomach.

experience. I have seen it only at the autopsy table.

Now we come to the chronic gastritides. Atrophic gastritis may require surgical therapy because of bleeding. In hypertrophic gastritis, as Dr. Clifton indicated, the surgeon may help to establish the diagnosis. Dr. Keller pointed out that it is very confusing to attempt differentiating either infiltrating or scirrhous carcinoma from malignant lymphoma. And, rarely, there may be surgical therapy for bleeding or obstruction. Last, granulomatous gastritis. We have done excisional therapy—partial gastrectomy—in one such case.

I have had no personal experience with tuberculosis of the stomach. It is now considered quite rare in this country. But syphilis of the stomach is still seen occasionally and should enter into our differential diagnoses whenever there is reason for suspicion on the basis of the patients' histories and from roentgen findings.

Let's take a few moments to consider some representative cases. Dr. Keller had the case of a man who awoke one morning and intended to take a drink out of the same bottle from which he had consumed a great deal the night before. Instead, he made the mistake of drinking about two ounces of concentrated acid. The man actually lived for seven years, and then died of a malignancy unrelated to gastritis. His injury required resection of nine-tenths of his stomach. He had only a little pouch left when we were through.

I might point out that surgical therapy is rather unsatisfactory in these unfortunate patients who have extensive corrosive damage to the stomach. In some of them, the surgeon makes a mistake in estimating the extent of damage, and in such instances leaks of the suture line are common. However, the commonest thing is that there are lesions that involve both the esophagus and the stomach. In dealing with the combined problem, we no longer use the jejunal interposition—i.e., resect the stomach and the lower end of the esophagus for corrosive damage. During the past two years we have done colonic interposition operations on two or more patients, and we feel that the new procedure is preferable. Most such patients are children who have had lye damage. The trend now is away from the use of the right half of the colon, and toward the substitution of the left portion of the transverse descending and upper sigmoid, and then restoration of colonic continuity, using the detached segments intrathoracically. Some are done through the anterior mediastinum, and some posteriorly. In children, we have tended toward the posterior approach.

Many of these patients are in deplorable condition nutritionally. Such was the case of the man who picked up the wrong bottle on the morning after the night before. He had lost a lot of weight; in fact, he was starving. In these cases it may be necessary to do a preliminary feeding jejunostomy.

Most of the lesions requiring surgical therapy are classified as cases of chronic gastritis. Various degrees of incredulity have been expressed by the other speakers regarding atrophic gastritis, and far be it from me to quibble with the cynics. I just don't know. Yet, I can't help feeling that the gastroscope is here to stay, and though it may now be an occasionally misleading diagnostic tool, we'll begin to use it more efficiently. There have been some oblique hints dropped here tonight that other lesions develop with abnormal frequency in association with gastroscopically-observed changes in the gastric mucosa.

Next, let's consider the case of a sick old man of 63 who had a polypoid mass that we felt was carcinoma, and on whom I did a rather generous gastric resection. The frozen sections were equivocal, and in the six tissue sections, indeed, the indications were no more than borderline as regards the diagnosis of malignant adenomatous polyps. He had striking gross changes of atrophic gastritis. The cardia of the stomach demonstrated the most severe changes, and bleeding began. We felt we had the field dry and sent him to his room, but he began to bleed worse as the hours went by, and the next morning I had to reopen the wound and, in an effort to stop the bleeding, I had to resect all of his stomach except for a narrow fringe around the esophageal opening in the stomach, estimated to be one or two centimeters in width. This bleeding from his atrophic mucosa resembled that of a fresh skin-graft donor site. It was that type of oozing. Nothing would stop it. We tamponed it, we put in Gelfoam and various local agents, and we tried cauterization, but it bled everywhere. This condition has been seen a number of times. We had a similar instance last summer. This is a terrifying complication of gastritis.

I should like to ask either Dr. Clifton or Dr. Towle whether this doesn't also occur occasionally after gastroscopy, in patients with severe atrophic gastritis. Also, how do patients behave when they have been subjected to the biopsy instrument to which Dr. Clifton has referred?

Dr. Clifton: It hasn't been a serious problem.

Dr. Tidrick: Well, anyway, the patient about whom I have been talking did live, and he is still living. He hasn't enough gastric mucosa left to run any great risk of cancer of the stomach, but he has a bit of a nutritional problem. When I last saw him about a year ago (it's now been about five or six years since his surgery), he was doing reasonably well.

Now atrophic gastritis and gastric polyposis. I'd like to present the interesting problem of a woman who haunted the medical outclinic at S.U.I. for years and came to be labelled a chronic psychoneurotic. She would tell us that she had vomiting, and she would come in and get a GI x-ray series. It would prove to be normal, and she would be sent

home with prescriptions for belladonna and various other things. Finally once, however, the radiologists caught an evanescent pyloric obstruction and an interesting mobile filling defect. She was prolapsing a gastric polyp in and out of the pylorus. That explained her symptoms.

Then, I should mention a patient whom Dr. Towle gastroscoped while he was a resident. He said that she had very severe chronic gastritis. I saw her eight years after the original gastroscopic examination, and she had a large, bulky, ulcerating neoplasm. Eight years after Dr. Towle gastroscoped her and made his comments about the severity of her chronic gastritis, this patient had associated pernicious anemia. She developed a thick, bulky carcinoma, and a radical resection was done, with a hemipancreatectomy, splenectomy and so forth. She survived for a year and a half, and then died of intracranial metastases.

A lady whom Dr. Clifton and I have followed for about eight years was 49 years of age when we first saw her. She had quite a large number of small polyps, and although on gastroscopic examination they appeared to be of the mucus retention type, rather than adenomatous polyps, we were disturbed. She didn't show atrophic gastritis at that time. I removed a strip of the greater curvature with a cluster of 20 or 30 polyps, and then cauterized 80 others, by actual count, using a short lighted proctoscope and an electrocautery unit. At follow-up gastroscopy just a few weeks ago, she showed a little recurrence. There were five or six small polypoid excrescences in the cardia, and there were some changes that suggested atrophy.

Speaking from a surgeon's standpoint, I find that the fascinating thing about these cases is the relationship between gastritis and the other things that can occur in the stomach—gastric polyposis and gastric carcinoma.

There is, of course, another difficulty, and that is the difficulty of diagnosis. A 61-year-old farmer who came to Iowa City recently had gastric x-rays which showed quite striking changes from those which had been taken five years earlier at another hospital. He had had symptoms for two months. Gastroscopic examination and gastric biopsy of the kind to which Dr. Clifton referred were done (by Dr. Clifton), and Dr. Keller and his colleagues saw him. They came to the conclusion that the patient might have a lymphoma, but they couldn't tell, and an exploration was done. Dr. Hickey explored him, and biopsied the mucosa. There were very prominent gastric rugae, but microscopically there were no evidences of serious disturbance.

I raise the question whether some of these cases that we loosely call hypertrophic gastritis shouldn't better be called benign gastric mucosal hypertrophy, and whether we shouldn't differentiate between the two.

GASTRITIS AND CARCINOMA

Dr. Towle: I wish to comment hurriedly on the three acute gastritides that I have seen during my experience as a gastroscopist, and to ignore completely the controversial hypertrophic gastritis. There are two points that I wish to make, and only two.

Of 444 consecutive gastroscopies performed in this county, 69 revealed the classical picture of chronic atrophic gastritis or gastric atrophy. Of those 69, polyps were present in four, bleeding in 17, and erosions in a great many. If I may use a category of Schindler's, "chronic superficial gastritis," which designates a condition that some people still believe is merely a precursor of chronic atrophic gastritis, I should like to report 41 cases so classified, with polyps in two more, bleeding in six and erosions in nine. My point is this, that 20 per cent of those gastritides—35 out of 175—were bleeding. I wish to emphasize that we see too many people with tarry stools and who have been bleeding, but whose GI series are negative or near-normal. The gastroscope thus has its place in determining the cause of bleeding, and certainly this is true in atrophic gastritis.

Polyps were found in seven of these 175, and polyps certainly are dangerous lesions in the stomach. If we include the group of gastritides occurring with other lesions, I saw 79 benign ulcers, and of them there were 11 that had occurred with atrophic gastritis. Atrophic gastritis with a benign ulcer still scares me.

Among these 444 gastroscopies 24 revealed carcinomas, and this point I think is important. Two of them were anaplasticas, so we'll make the number 22. With those 22 carcinomas of the stomach, atrophic gastritis of the stomach was concurrent in 21 cases. Now I'm not drawing any conclusions from those figures. There was one recurrent carcinoma in the suture line that also was accompanied by atrophic gastritis or gastric atrophy. There are three things, then. Bleeding occurred, particularly in atrophic gastritis, and hemorrhage occurred. Polyps appeared, and the bedfellow of carcinoma of the stomach, in my opinion, is atrophic gastritis.

I should like to have Dr. Clifton comment on the so-called pre-pyloric lesions. No one has mentioned prolapse of the gastric mucosa, though silence on that point is probably a good thing. And I should like to have Dr. Clifton comment on the coexistence of atrophic gastritis with carcinoma.

Dr. Clifton: Biopsy studies that other men have performed have destroyed many of my notions about gastritis. I hasten to emphasize that my personal experience with biopsy of the stomach has been very limited. My work with the Rubin instrument has been confined to the small intestine, and I don't have a great deal of personal experience to support my impressions.

If we accept as fact that morphology (of specimens secured through biopsy) is the basis of therapy, then it seems to me that we must readjust our thinking about gastritis. Of course biopsy can produce erroneous results. Certainly there are possibilities for error in cases of patchy gastritis. Obviously, one can't always be sure that he has biopsied the exact area where he thinks he has seen the pathology.

Doig and Wood studied this problem of sampling error, as well as they could, and have come to the conclusion that the chance of error is in the neighborhood of four per cent.

For years, I diagnosed gastritis on the basis of gastroscopy, and I think all of us were confident of such diagnoses until the biopsy studies began coming out. Now, however, I am always suspicious that what I think I'm seeing really *isn't* there, and what I'm not seeing *is* there. As I said before, I believe that in gastric atrophy and in severe superficial gastritis, the gastroscopist is correct most of the time. For the shades in between, I think he may frequently be wrong, and in the matter of hypertrophic gastritis, I am sure that in the past he was entirely in error.

Now I don't know what we clinicians should do about people with x-ray negative dyspepsia. I should certainly like to see someone like Dr. Towle, whom I know to be an expert gastroscopist and who is practicing where he sees a great many such patients and can follow them—do biopsies on such people. In a place like University Hospitals, we get a biased or slanted view of this disease, as we do of most diseases. In brief, I think we need simply to be careful with our diagnoses, and try to substantiate them whenever possible.

In regard to the prepyloric suspect lesion, I have felt that gastroscopy is fine, if it showed the lesion in the prepyloric area. But if the lesion can't be seen, I have been in the habit of simply ignoring the gastroscopic findings and going ahead with gastric cytology, with radiology and, if need be, with surgery.

Prolapse of the mucosa, again, is a difficult topic. I am convinced that it is a definite entity—that it does occur. But once again I don't believe it has been conclusively shown to produce a certain cluster of symptoms. In most people it is asymptomatic. I also believe that it will bleed on occasion.

I too am frightened by a gastric ulcer, even though it appears to be benign, when it occurs in association with atrophic gastritis. It is one of the things that inclines me to advise the patient to have surgery. However, biopsy studies by others have shown that atrophic gastritis is a frequent accompaniment of benign peptic ulcer. Perhaps we have been unduly concerned by the gastroscopic appearance of atrophic gastritis in association with ulcer.

I hope that no one gets the idea that I am a complete nihilist as regards gastritis. It obviously exists, and it obviously produces symptoms in some people, even though it just as obviously doesn't produce symptoms in a great many others. Thus, it is difficult to say that there is a specific clinical syndrome that results from it.

Dr. Towle, I haven't been able to get my colleagues at University Hospitals to refer bleeders quickly so that I might gastroscope them. And I have been amazed at the paucity of bleeding gastritis that I, as a gastroscopist, have seen. This is due to the fact that I never get to see these people until some days after they have had x-rays and until four or five staff men have been consulted and have finally suggested gastroscopy. By that time, seven to ten days have elapsed.

Dr. Towle: Where would allergic gastritis fit into the classification that has been presented here tonight?

Dr. Clifton: I'm not sure, but in the literature there is an account of a study that was done on a person who presented proved sensitivity to certain allergens. The allergens were deposited upon the gastric mucosa, and the individual was then gastroscoped at intervals. The authors described a number of reactions very beautifully—hyperemia, eczema and sometimes petechial hemorrhages.

Dr. Towle: I'd like you other men to express an opinion about these mild cases of gastritis that we have seen with the gastroscope, for I think you have had an opportunity to follow those people over a period of time. I'd like to hear your opinions about the correlation of symptoms and what we see through the gastroscope.

Dr. Tidrick: Through the gastroscope, I have seen hypertrophic gastritis patients hemorrhage, but those people may have had the giant hypertrophic gastritis that remains in the classification. I have had opportunities to gastroscope many people more than once, and whether repetition of the procedure had something to do with the severity of their symptoms, I don't know.

I have seen hypertrophic gastritis disappear in 72 hours, though the cobblestoning had been 3-4 in. in diameter. Thus, I take as dim a view of this lesion as you do, Dr. Clifton. I have seen a benign ulcer disappear in five days—one that had measured about 2 cm. in diameter. On the other hand, I think that the patient with atrophic gastritis has specific symptoms, and I think that the one with giant hypertropic gastritis has some too. These patients bleed and have polyps. I think they should be watched.

Let me say that I don't think the diagnosis of chronic hypertrophic gastritis has been warranted nearly as frequently as it has been offered during the past 15 years.

Dr. Tidrick: Reference has been made to obstruction in association with hypertrophic gastritis, or with benign mucosal hypertrophy, as the case may be. It does exist, for I have seen it at least once, where there was prolapse through the

pylorus, and where pyloroplasty apparently cured the patient. Occasionally, invagination or a form of intussusception occurs. I have had no personal experience with that complication.

I wonder, Dr. Towle, whether you have set the incidence of malignant change in association with atrophic gastritis high enough. I don't think we have sufficiently emphasized that the patient with established atrophic gastritis has a sword of Damocles hanging over him. In other words, I think that Dr. Towle's estimate that the cancer incidence has increased four-fold is a bit on the modest side.

Dr. Towle: I think I said that among my 23 carcinoma patients, 21 had atrophic gastritis with their cancers, and I gave the percentages of my cases in which there were polyps and in which there was bleeding. As regards the incidence generally, all I can say is that the frequency of carcinoma with atrophic gastritis has stepped up to, I think, better than 4.8 per cent, which represents a very considerable share of total hospital admissions.

Dr. Braatelein: I should like to ask Dr. Towle and Dr. Clifton whether they know of a study that has shown the correlation between actual biopsy, diagnosis through the gastroscope, and the subsequent development of carcinoma.

Dr. Clifton: You mean where the diagnosis of atrophic gastritis was established on a given day by biopsy, and then some months later cancer developed? I don't know of such a study.

Dr. Towle: I know of several patients to whom that sequence has occurred, and I know of one doctor here in the room who will bear out my statement, for it happened to his father.

IATROGENIC PERFORATION

Dr. Towle: Another question: What is the incidence of perforation with gastroscopy? I can say only that I have been extremely lucky, and the last time I talked with Dr. Schindler he asked me whether I'd had any gastroscopic accidents. When I said I had had none and had told him how many I had done, he said, "You're overdue." Since then, I have had one perforation out of some 450 gastroscopies. The man did very well after surgery. Out of the first 500 done at Iowa City, we had three perforations. In one instance, we didn't know whom to blame. The patient had been bronchoscoped and esophagoscoped on successive days.

Dr. Clifton: I don't have any particular figures, Dr. Towle. It's not that I am hiding anything, but as you know, in our residency program the residents actually do the instrumentation after I have shown them how to do it. Thus, the actual number of times that I have personally passed the instrument down the esophagus is not very great. I have been in charge of the gastroscopy program at University Hospitals since 1953, and during that time I suppose we have done roughly 2,000 procedures. About 5,700, all told, have been done at University Hospitals, of which 3,500 had been done before I took charge. In my seven-year period we have had two perforations that I can remember off hand. One occurred last year, and the other about five years ago.

I think that, by and large, gastroscopy is a very safe procedure. Like anything else, one must learn to perform it, and in the learning process one doesn't do as good a job as he will do 10 years later.

The glass fiberscope which has been put on the market by the American Cystoscope Makers, Inc., is sure to revolutionize gastroscopic work. It is absolutely flexible, for it can be tied into a knot, and I anticipate that perforation will be as rare with this instrument as it is with an Ewald tube. Furthermore, it will eliminate many of the blind areas and will let us visualize the interior of the duodenal bulb.

Flu Vaccinations for High-Risk Groups

The Public Health Service announced on October 22 that very little influenza had as yet been reported around the nation, but emphasized the importance of using available supplies of influenza vaccine to immunize persons in the high-risk groups before the onset of winter.

Those to whom vaccination is most important include: (1) persons of any age who suffer from chronic diseases of the heart, lungs and circulatory system; (2) pregnant women; and (3) persons over 65 years of age. Between September, 1957, and March, 1960, people in those three groups accounted for most of the 86,000 influenza-related deaths.

Although vaccine has been in short supply in many areas in recent weeks, the PHS anticipates that there will be plenty to immunize the high-risk groups before the end of the year. In times of shortage, this protection may be limited to only one injection, since it is the first shot that gives the most immunity. Until supplies become ample, vaccine should be limited to the high-risk groups.

Vaccine manufacturers have produced more influenza vaccine during the past three months than in all of the preceding year.

Some increase in local outbreaks of two types of influenza—A and B—has been forecast by influenza experts for this season, the forecast being based on observed influenza cycles. Type A tends to occur in two or three year cycles and has been dormant in this country since 1960. Type B generally occurs in four to six year cycles and hasn't been prevalent in this country for more than six years. Both types were prevalent in various countries last year, notably in England and Japan.

Hemorrhage From a Gastric Leiomyoma During Anticoagulant Therapy

Report of a Case

NORVAL L. SAXTON, M.D.
CEDAR RAPIDS

THERE HAVE BEEN few, if any, reports in the literature regarding hemorrhage from a leiomyoma of the stomach in which the bleeding began as a consequence of anticoagulant therapy. The patient whose case is to be described here started to bleed from the gastrointestinal tract after 24 days of that type of medication. Roentgenograms of the barium-filled stomach, taken on admission, had suggested a benign lesion of the gastric wall consistent with gastric leiomyoma, and thus it was suspected as the source of the bleeding. Subsequent surgery and histologic examination confirmed that the bleeding indeed had originated in a gastric leiomyoma.

CASE REPORT

A 60-year-old man was admitted to the hospital on January 20, 1960, because of severe substernal chest pains that had begun one month earlier. He had been seized on December 17 and 18 by severe, "pressing" substernal pains that radiated into the neck and left arm, and which disappeared in less than 30 minutes. Neither attack had been associated with exertion. One week later, he had begun to have frequent similar, but less intense episodes of pain six to 10 times per day, and relieved them by taking the nitroglycerin that his local physician had prescribed. He described a premonitory symptom consisting of "an odd taste" in his mouth. He had then been hospitalized in his home town for 15 days for cardiac evaluation, and a diagnosis of myocardial insufficiency was made. Two days after his discharge from the hospital, he was readmitted because of a sudden loss of appetite, bloating unrelated to meals, and a peculiar "rolling sensation" felt under the left rib margin. He denied vomiting, nausea, eructations, pain or melena. Upper intestinal radiographic studies revealed what appeared to be an intramural gastric lesion. On the day of a planned exploratory laparotomy, he was again seized by severe substernal pains, and was subsequently referred to University Hospitals, Iowa City.

The patient had noticed increasing shortness of breath on exertion over the previous year, and had coughed up blood-tinged sputum on a few occasions two weeks prior to admission. He denied orthopnea, paroxysmal nocturnal dyspnea, ankle edema or other cardiac symptoms, and likewise denied recent weight loss or gastrointestinal symptoms prior to the episode that had led to his second hospitalization.

Physical examination revealed a well developed, cooperative man in no distress. The fundi were normal, with the exception of grade I arteriosclerotic changes. The lungs were clear. The point of maximum cardiac impulse was in the fifth intercostal space and midclavicular line. There was a regular sinus rhythm. No murmurs or rubs were heard. The abdomen was scaphoid. There were no abnormal masses, and the liver was not enlarged. There were bilateral, easily reducible inguinal herniae. The rectal examination was negative. The extremities were normal. Peripheral pulses were equal and active throughout. There was no evidence of cyanosis of the skin. The neurologic examination was normal.

The blood pressure was 134/90 mm. Hg, the pulse was 80, the respirations were 24 per minute, and the temperature was 100.8°F., orally.

The urine was negative for albumin or sugar, and the sediment contained two to five white blood cells per high-power field. The specific gravity was 1.026. A repeat urinalysis during the patient's hospital stay was normal. An examination of the blood revealed a hemoglobin of 13.1 Gm./100 ml., and a white cell count of 12,600, with 70 per cent neutrophils, 25 per cent lymphocytes and 3 per cent monocytes. A serologic test was negative. The blood urea nitrogen was normal. A Meyer's test for blood in the stool was negative. An electrocardiogram taken on the day after admission showed a regular sinus rhythm, with a ventricular rate of 68. ST and T wave abnormalities suggested myocardial disease.

The patient was placed at bed rest, and was given nitroglycerin sublingual tablets as needed, for precordial pain, and long-acting Peritrate.

Three days after admission, the patient developed a sudden deep thrombophlebitis of the left leg, manifested by pain, marked swelling and increased skin temperature of the affected extremity. Anticoagulant therapy was begun immediately,

Dr. Saxton is an interne at Mercy Hospital, Cedar Rapids. He wrote this paper before taking his medical degree at S.U.I. last spring, and he wishes to express his thanks to Dr. John A. Gius, of the Department of Surgery there for giving him permission to write a report of this case and for encouraging him in presenting it for publication.

with 50 mg. subcutaneous heparin every eight hours and an initial dose of 50 mg. of Coumadin. The baseline prothrombin time was 15.8 seconds. On the second day, the prothrombin time had increased to 23 seconds, and 5 mg. of Coumadin was given.

On the next two days, the prothrombin times were 39 and 50 seconds, respectively, and only 10 mg. of Coumadin was given during that time. During the following two days, the prothrombin time dropped to 28.8 seconds, and was maintained between 29 and 25 seconds by means of doses averaging 2.5 mg. until February 14 (a total of 24 days of anticoagulant therapy). Then the patient began to hemorrhage. Melena was noted, and the patient exhibited profound weakness. Coumadin therapy was stopped, 5 mg. of vitamin K_1 was given orally, and the prothrombin time promptly dropped to 17.1 seconds on the following day. The patient's hemoglobin had decreased to 5.6 Gm./100 ml., and eight units of whole blood were given him over the next three days. The hemoglobin returned to 13.1 Gm./100 ml. four days after the onset of the bleeding. The stools became lighter seven days after the onset of the hemorrhage, and the Meyer's test for blood was 1+ to 2+.

The patient was transferred to the Department of Surgery on February 19, and was considered in satisfactory condition at that time. Gastric secretion studies were negative for free hydrochloric acid and were positive after the administration of histamine. Gastric aspirate at that time contained blood. The left leg was still swollen, but there was no pain and no temperature difference between the two legs. Sigmoidoscopy to 14 cm. was normal.

On February 26, the patient underwent an exploratory laparotomy via an upper bilateral transverse incision. A round and nodular mass 2.5 cm. in diameter and 12 Gm. in weight was found on the greater curvature in the dependent portion of the antrum of the stomach. The lesion was freely movable, and there was no gross evidence of extension. A frozen histologic section was judged borderline between a leiomyoma and a leiomyosarcoma. There was no evidence of metastasis to regional nodes or to the liver.

The nodule was excised, along with a 1 cm. margin, and the stomach was closed. Later histologic examination was felt to be consistent with benign leiomyoma.

The patient's postoperative course was complicated by frequent vomiting, anorexia and fluid-electrolyte imbalance. Intravenous maintenance was required for 11 days. It was felt that the patient had anatomical closure of the gastric outlet.

On March 9, the patient again underwent abdominal exploration via the previous incision. The area of the leiomyoma excision was examined, and tight closure of the gastric lumen, secondary to edema, was found. A 50 per cent gastric resection, a gastroduodenostomy, and a feeding jejunostomy were carried out. Adhesions in the area of the first incision were responsible for accidental entrance into the small bowel. An end-to-end anastomosis was carried out, after 4 cm. of jejunum had been resected.

The patient's postoperative course was uneventful, and he was discharged on March 20 and given nitroglycerin for the continuing anginal pains.

DISCUSSION

Serious or fatal hemorrhage from gastric leiomyomata is not a common occurrence, because of the fact that gastric smooth muscle tumors account for such a small percentage of the clinically significant gastric tumors.[1] Hemorrhage is the most frequent complication of this tumor and of its malignant counterpart, the leiomyosarcoma. The sarcoma will not be discussed here.

With the increasing use of anticoagulant therapy for myocardial infarction, deep thrombophlebitis, cerebrovascular disorders and peripheral occlusion, the contraindications and complications of such therapy become increasingly important.

Hemorrhage is the chief complication of anticoagulant therapy, and may occur in from 2 to 19 per cent of patients receiving prothrombinopenic agents. The sites of bleeding are most frequently the skin, the genitourinary tract and the gastrointestinal canal.[2] It is obvious that any preexisting lesion capable of hemorrhage assumes a greater importance during this type of therapy. The gastric leiomyoma is one such lesion. Its tendency toward ulceration[3] and hemorrhage can produce a confusing and catastrophic picture during anticoagulant therapy because of the frequency with which the lesion may be present but totally unsuspected.

There are few absolute contraindications to antithrombotic therapy. They are actual bleeding or the presence of significant hemorrhagic diathesis.[4] The other contraindications are relative, and are arrived at by weighing the benefits of the therapy against the possible dangers in each individual case. Close observation is mandatory for all patients who are on this type of therapy. Should bleeding occur from some site during treatment, one need not conclude that the agent should be withdrawn. Rather, it is a definite indication for careful evaluation of the patient, and a careful search for a previously unsuspected lesion or disorder. The literature contains reports of many unsuspected gastric carcinomas, peptic ulcers, polyps, etc. that began to bleed when the patient was given anticoagulant medication. It is possible that any benign or malignant process, especially one with a tendency toward ulceration, might become manifest and life-threatening in this manner.

Smooth muscle tumors, of which leiomyomas are the commonest, make up only one per cent of all gastrointestinal neoplasms.[5] Gastric leiomyomas account for between two and four per cent of all clinically significant gastric tumors.[1] Slowly accumulating information suggests that the inci-

dence of leiomyomata in the gastrointestinal tract, and especially in the stomach, is far greater than was previously thought. Meissner reported an incidence of gastric leiomyomata in 46 per cent of 50 random autopsies.[6] Subsequent investigators have reported figures ranging from 10 to 50 per cent. The presence of these lesions in otherwise normal individuals is rarely suspected, perhaps because of the size of most of the tumors. Most of the incidentally found and asymptomatic smooth-muscle tumors are less than one centimeter in diameter.[6] Even the large tumors of this type may be entirely asymptomatic and remain undiagnosed until necropsy. Diagnosis of these neoplasms on the basis of their non-specific and variable signs and symptoms is notoriously inaccurate. In the absence of diagnostic roentgenologic signs, their frequent symptoms of mild dyspepsia and anemia, especially in the older age groups,[7] are often overlooked. The commonest symptom and complication of the gastric leiomyomas is hemorrhage—minute or massive.[1, 3, 5, 7, 8] Less frequent symptoms, in the order of their appearance, are pain or discomfort, palpable mass, weight loss, weakness, and symptoms simulating peptic ulcer.[1] The presence of these smooth-muscle neoplasms, silent or manifest, becomes more important when anticoagulant therapy is instituted for a cardiovascular or cerebrovascular disorder.

The leiomyoma found in the case that I have described was not extremely large, and had caused the symptoms of bloating, a "rolling sensation" beneath the left rib margin, and anorexia for a period of less than one week before hospitalization. These symptoms are typical of the extraordinary and confusing picture often presented by the benign leiomyoma. This patient had no history of previous bleeding from the alimentary tract, and entered the hospital with a normal hemoglobin.

Unlike the bleeding of peptic ulcers, the bleeding of these tumors is more often severe than occult, and Highman thinks that the chance of their hemorrhaging is a greater hazard to the individuals who have them, than is the chance that they are malignant.[7] This may be accounted for by the fact that these tumors have a tendency to ulcerate, and the usual small ulceration of the mucosa is typically located at the site of attachment of the tumor to the gastric wall. The vessels supplying the relatively avascular tumor are more numerous

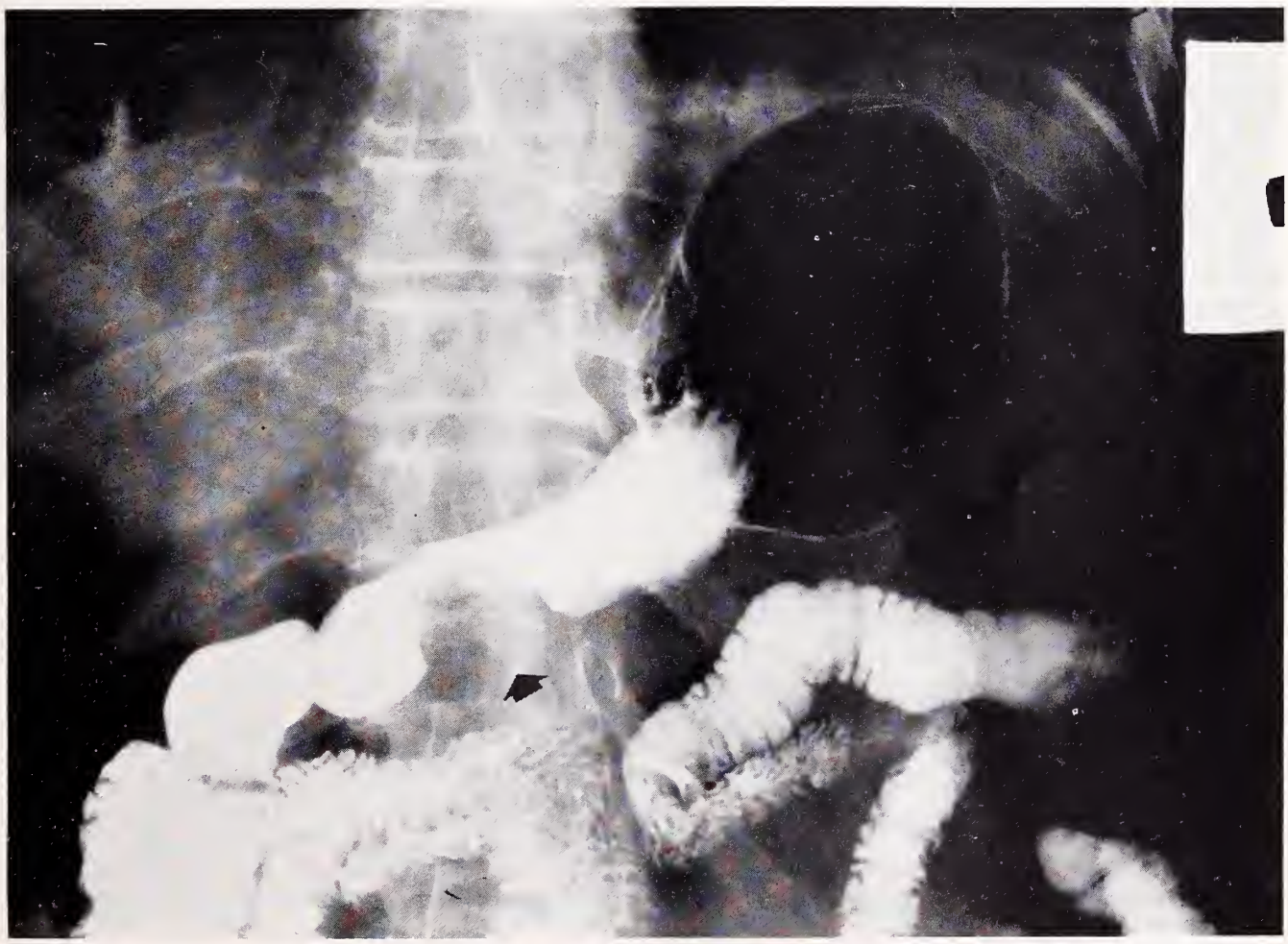

Figure 1. Roentgenogram showing filling defect in the pars media along the greater curvature. The defect is smooth and appears to be intramural.

and larger at that site, and thus a large volume of blood is lost through this small ulcerated area when the vessels erode.[9] Marshall and Meissner reported that the ulcerations are often deeper than those seen in the peptic ulceration.[10] I have recently seen such an ulceration in a leiomyoma less than two centimeters in diameter that had been removed from a young boy. Its depth was approximately one centimeter.

Roentgenographic evidence of a gastric-wall tumor was found in the case that I have described earlier in this paper, at the time the symptoms were first noted. The films were interpreted as showing an intramural lesion consistent with a leiomyoma, neurilemmoma or pancreatic cell rest. On the basis of statistics, a leiomyoma was considered most likely. The preoperatve diagnosis is usually not so simple, and the symptomatic tumors are frequently not to be seen on the roentgenogram. When the diagnosis can be made by this technic, it may be quite characteristic. The classic roentgenologic characteristics are usually as follows: a well defined, rounded filling defect in the stomach filled with barium; good tumor mobility; smooth mucous membrane covering the defect; normal surrounding mucosa; normal peristalsis;[11] and a split stream of contrast medium as it passes over the tumor.[12] The presence of these signs doesn't necessarily mean that a leiomyoma will be found at surgery, however, for other benign or malignant lesions can occasionally mimic this picture.

The absence of the above signs does not rule out a benign lesion, for many large and symptomatic tumors have been missed by the roentgenologist. Some of the tumors are extramural—a circumstance that makes the diagnosis more difficult. In spite of its shortcomings, however, roentgenography is the best diagnostic tool we have for this purpose. Gastroscopy has been helpful with some of these tumors, and its real value in this type of tumor diagnosis has yet to be fully assessed.

At the time our patient developed deep thrombophlebitis, we began anticoagulant therapy with heparin and Coumadin (warfarin-sodium). Coumadin is now regarded by many physicians as the most nearly ideal of the prothrombinopenic agents.[4] The patient's prothrombin times were easily controlled through the administration of small doses of Coumadin (1.25 to 2.5 mg./day), and there was no variation or alteration in either the prothrombin time or the patient's condition prior to the onset of his massive gastrointestinal bleeding. We were at a loss to account for the initiating factor of the hemorrhage. In spite of the characteristic roentgenologic picture that the tumor presented in this instance, there was no ulcer niche clearly evident (Schindler's sign),[1] although one radiologist thought a niche was suspect in the films. Yet, a central deep ulceration was present at operation. The ulceration may have developed during the month of anticoagulant therapy. The bleeding nearly ceased within 24 hours after the anticoagulant was stopped and vitamin K_1 was given. Whole blood was administered, and within four days the hemoglobin level was back to normal.

When the patient's condition was felt to be satisfactory, his abdomen was explored. A submucous leiomyoma with central ulceration was found on the greater curvature in the antrum of the stomach. Submucosal location is twice as frequent as is subserosal. The lesions are found along the lesser curvature and posterior wall twice as frequently as on the greater curvature and posterior wall. They have little predilection for a specific division of the stomach.[1]

Wedge resection with a 1 cm. margin was the treatment of choice in this case, but most surgeons prefer subtotal gastrectomy. The reasons most commonly given for the more extensive procedure are chiefly concerned with the possibility of metastasis in the event the tumor is a leiomyosarcoma. Frozen histologic sections of these tumors are difficult to interpret, for the microscopic difference between the two tumors may be slight. Ideal histologic preparations are not infrequently confusing, so that the diagnosis often depends upon the pathologist's experience and attitude.[1] Postoperative closure of the gastric outlet, in our patient, necessitated re-entry and a subtotal gastrectomy. This complication, or the likelihood of its occurrence, doesn't dictate the use of the more extensive surgical procedure, but the sparse statistics on metastasis of the sarcoma (between 15 and 25 per cent[1, 8]), mainly to the liver, and the histologic similarity of the benign and malignant tumors may be considered adequate reasons.

Although this case demonstrated bleeding from a gastric leiomyoma, spontaneous hemorrhage can occur from benign tumors or lesions in other parts of the gastrointestinal tract when the patient is receiving anticoagulant therapy. Other benign but less frequently appearing tumors that are capable of bleeding include adenomas, lipomas, pancreatic cell rests and neurilemmomas. Benign tumors along the entire length of the gastrointestinal tract are potential sites of hemorrhage, and the possibility of bleeding increases under the influence of anticoagulants. The size of the tumor seems to have no relation to the likelihood of its bleeding.

Fatal hemorrhage from a benign tumor is rare, but it can and does occur. Whether mild or severe, each such episode of bleeding deserves careful watching and a search for its source.

Few surgeons encounter leiomyomas or any other benign tumors of the stomach with sufficient frequency to become completely familiar with them. Because of their commonly asymptomatic nature, their tendency to ulcerate and hemorrhage, and the increasingly common use of anticoagulants, physicians and surgeons should be aware of the leiomyoma's potentialities.

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State University of Iowa College of Medicine

Clinical Pathologic Conference

SUMMARY OF CLINICAL FINDINGS

A 42-YEAR-OLD shop foreman was admitted to the hospital in coma, on April 22, 1961, and his history was obtained from his wife. The patient had been ill, off and on, since September, 1960. His first symptom had been a non-productive cough. On January 12, 1961, he had been hospitalized because of the cough, a 25-pound weight loss and night sweats. During that period of hospitalization, he had had a transient episode of slurred speech. A chest x-ray at that time had shown a left upper lobe infiltrate, skin tests had been negative for tuberculosis, and the sputum had been negative for acid-fast bacilli. No cultures or guinea pig inoculations had been done. The diagnosis at that time had been pneumonia, and the patient had been treated with antibiotics. He had then improved, and he had been feeling better when his family doctor saw him on February 3, and a chest x-ray showed regression of the infiltrate in the left upper lobe. Because the lesion hadn't cleared, he was given 1 Gm. of streptomycin per week up until the time of his admission to this hospital.

During March he had been ill again with fever and increased coughing. A chest x-ray taken on April 3 had shown no change from the one taken on February 3. On April 12, he had been hospitalized because of fever, mental confusion and night sweats. On April 17, he had coughed up a large amount of greenish sputum, and he had then been treated with many antibiotics without improvement. On the morning of April 22. the day of his admission here, he had been able to take liquids by mouth. Throughout the three months prior to his entering this hospital, he had blurred vision and had "to read slowly."

Physical examination revealed a comatose, moderately dehydrated man whose rectal temperature was 102°F. His respirations were 52 per minute, his pupils were 5 mm. and 3 mm. in diameter, and neither reacted to light. The right tympanic membrane was scarred, but the left appeared normal. The mucous membranes of the mouth were dry. The neck was 4+ rigid to antiflexion. The chest was symmetrical. Expansion was fair, dullness was present over the left upper lobe, and the breath sounds were diminished over the left upper lobe. Some rhonchi were heard over the right upper lobe.

The blood pressure was 200/80 mm. Hg. The ventricular rate was 84 per minute, and the rhythm was regular. The left border of cardiac dullness was at the midclavicular line. No murmurs were heard. The abdomen was flat, and the soft organs were not palpable. There was no edema of the ankles. The deep tendon reflexes were active and equal. The Kernig and Brudzinski tests were positive. The plantar reflexes were bilaterally flexor. The patient responded to painful stimuli by withdrawal. No enlarged lymph nodes were felt. Some of the time he had Cheyne-Stokes respiration.

No urine specimen was obtained. The hemoglobin was 13 Gm., and the white count was 34,300/cu. mm., with 83 per cent segmented polymorphonuclear leukocytes. The spinal fluid pressure was greater than 600 mm. H_2O, and there were 19,300 cells/cu. mm., mostly polymorphonuclear leukocytes. The spinal fluid sugar was 0 mg./100 ml., the chlorides were 107 mg./100 ml., and the protein was 856 mg./100 ml. The spinal fluid was cloudy, and a pedicle [sic] formed after the tube had stood for several hours. A spinal fluid smear was negative for acid-fast bacilli. Gram's stain showed many gram-positive cocci in chains. The blood sugar was 150 mg./100 ml., the blood urea nitrogen was 7.0 mg./100 ml., the creatinine was 0.9 mg./100 ml., the CO_2 combining power was 22 mEq./L., the sodium was 136

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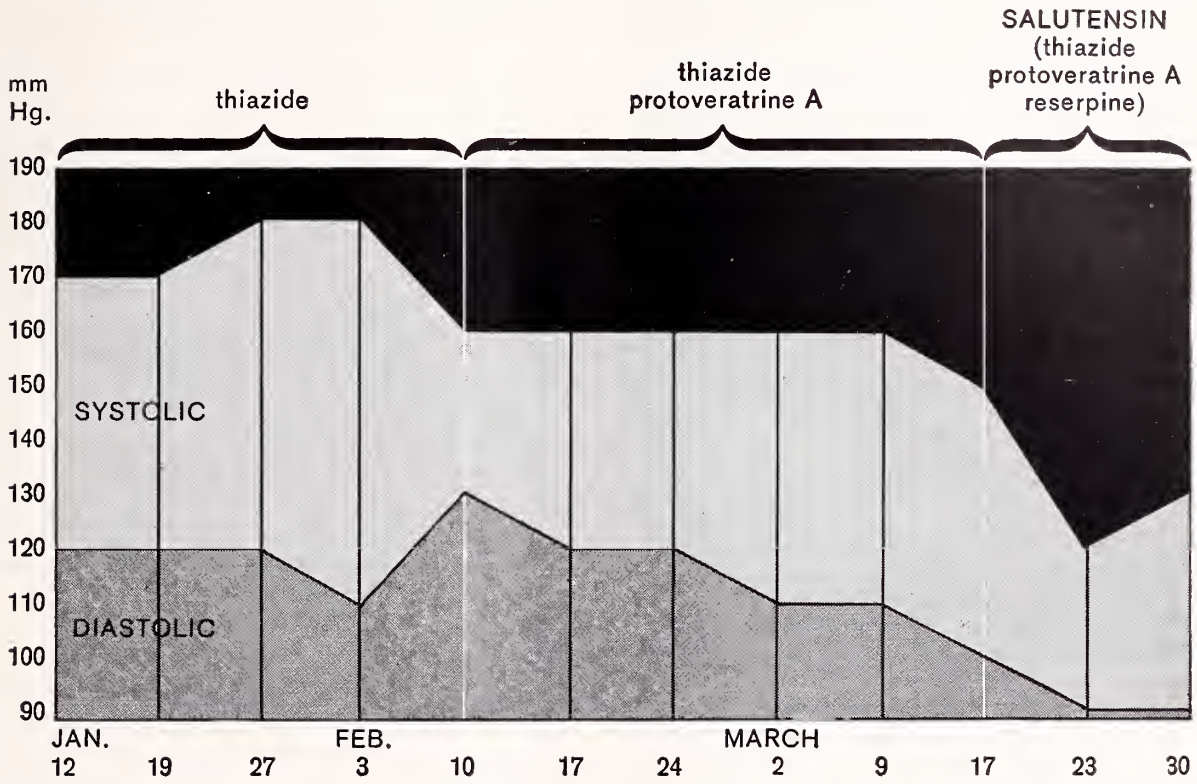
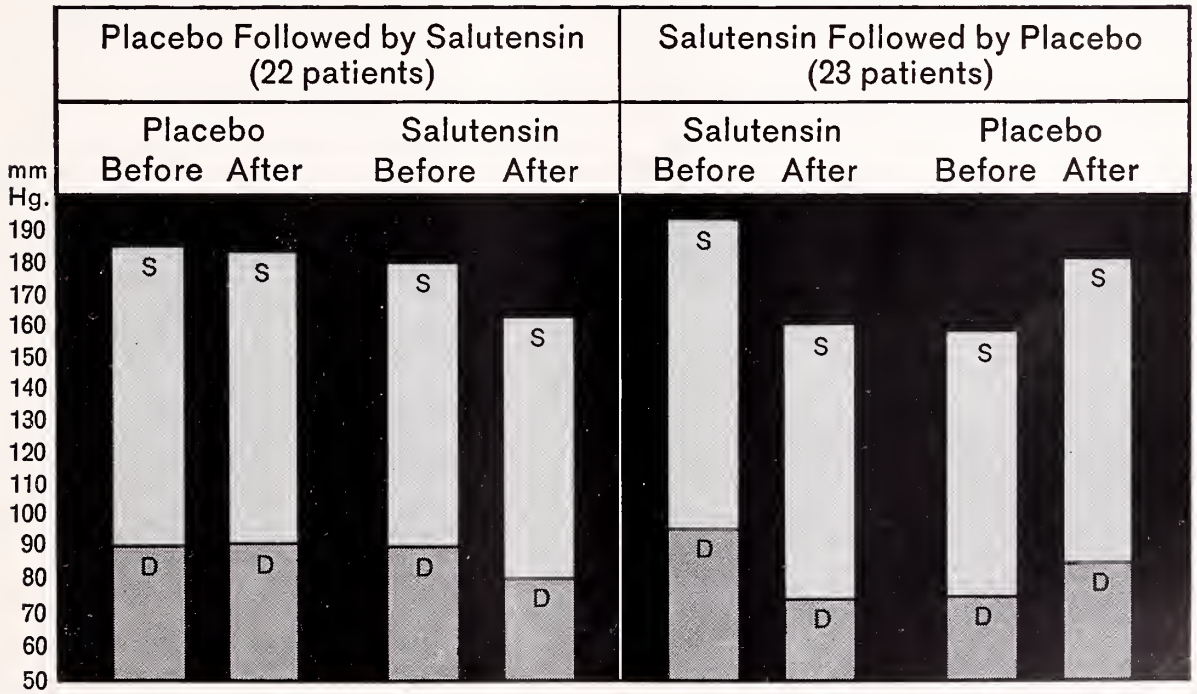
11 WEEKS TO LOWER BLOOD PRESSURE TO DESIRED LEVELS BY SERIAL ADDITION OF THE INGREDIENTS IN SALUTENSIN IN A TEST CASE

(Adapted from Spiotta, E. J.: Report to Department of Clinical Investigation, Bristol Laboratories)



3½ WEEKS TO LOWER BLOOD PRESSURE TO DESIRED LEVELS USING SALUTENSIN FROM THE START OF THERAPY IN A "DOUBLE BLIND" CROSSOVER STUDY

Mean Blood Pressures–Systolic (S) and Diastolic (D)



In this "double blind" crossover study of 45 patients, the mean systolic and diastolic blood pressures were essentially unchanged or rose during placebo administration, and decreased markedly during the 25 days of Salutensin therapy. (Smith, C. W.: Report to Department of Clinical Investigation, Bristol Laboratories.)

BRISTOL LABORATORIES/Div. of Bristol-Myers Co., Syracuse, N.Y.

mEq./L., the potassium 3.9 mEq./L., and the chloride was 96 mEq./L.

After blood and bone marrow cultures had been obtained, treatment was started with the following drugs: hydrocortisone, 100 mg. I.V.; streptomycin, 1 Gm. I.M.; Isoniazid, 100 mg., subcutaneously; aqueous penicillin, 1,000,000 units every three hours I.M.; tetracycline, 500 mg. I.V.; and sodium sulfadiazine, 5 Gm. in 1,000 cc. saline I.V. The patient continued in poor condition and died 12 hours after admission.

SUMMARY OF CLINICAL DISCUSSION

Dr. George N. Bedell, Internal Medicine: The CPC for today concerns a 42-year-old shop foreman who had been well until September, 1960, and who died in April, 1961. Because of cough, weight loss, night sweats and an abnormal chest x-ray, a diagnosis of pneumonia was made in January, and the patient was treated with antibiotics. He improved, but became ill again during March and April. He eventually developed signs of central nervous system involvement, as well as pulmonary involvement.

Dr. Bean is going to discuss the case.

Dr. William B. Bean, Internal Medicine: As I sometimes do, I shall introduce my comments with some verse:

You all can see with valid reason
We're in the open CPCeason.

Since I'm not fixed like television,
My errors may require revision.

Pathologists use every free trick
To make the speaker CPCeasick.

The protocol is chart and compass,
But CPC'ing naught will bump us.

No fickle wind will wreck our sails
If CPCeamanship prevails.

Red herrings mimic real disease
In all the treacherous CPCeas.

Some data may be deleterious,
Not to be thought as CPCerious.

But other data raise the curtain
Suggesting what is CPCertain.

Though if your mind works clear and freer,
You'll rank high as a CPC'er.

For CPCecrets understood
May lead to answers true and good.

If I don't come out very well,
I'll put the blame on George Bedell

Who wrote the protocol for me
To drown me in the CPCea.

If he should be gift-bearing Greek,
To him I'll turn the other cheek.

So minding every "Q" and "P,"
Let's sail upon this CPCea.

The protocol, I must point out, contains what I trust is a misprint. At somewhere near the middle of the next-to-last paragraph, we are told, "A pedicle formed. . . ." Those of us who have studied Latin know that a pedicle is a little foot. It would have been most extraordinary for a little foot to form in a tube of spinal fluid. What was meant, I am sure, is that a pellicle—a little skin—formed. Now, having delivered myself of my academic obligation, I shall address myself to the patient's clinical problem.

Dr. Bedell has reviewed the highlights and low marks of our patient's sad pilgrimage to his death, but I shall go over the information piece by piece, for I am uncertain and confused by the various findings, each one of which tends to exclude a diagnosis that might explain his difficulty. I'll share my confusion with you.

Eight months before our patient died, he started having a non-productive cough. There was no statement about his smoking habits. If this man had cancer of the lung, smoking might have been mentioned either as a clue or as a red herring. Added to the cough were night sweats and a significant loss of weight. Over a period of eight months, his loss of 24 lbs. means that he was in a negative calorie balance of somewhere around 3,000-4,000 calories per week. He probably wasn't eating well during part of that time, but the fever and the increased metabolism accounted for part of his weight loss.

He then had a period of slurred speech, and he also had blurred vision and read slowly. These occurrences suggest some serious process deranging the function of his brain. A lesion in the brain, however, would have had to cover a great deal of ground to produce so many different kinds of things.

Dr. Keller, do you have the x-rays of the early or of the late stages?

Dr. John Keller, Radiology: We have both.

Dr. Bean: First, we must get the patient into the hospital, and then we can have Dr. Keller tell us about the x-rays.

At the time the man was first examined, there was a left upper lobe infiltrate. The skin tests were negative, and assuming that the tuberculin material was adequate and that the tests were done properly, the results constitute strong testimony against tuberculosis as an explanation for all of his trouble at that moment. However, tuberculosis is sometimes inactive. Sometimes too, one gets an "unexplained" false negative. The anergic state that occasionally prevails in overwhelming tuberculosis might explain a negative tuberculin test result at this stage of his illness.

No acid-fast organisms were found in his sputum. There were no cultures or guinea pig inoculations. He was thought to have pneumonia, and was treated with antibiotics. He improved. When his family doctor saw him some time later, he was feeling better. The x-ray at that time showed regression of the infiltrate in the left upper lobe, but because the lesion hadn't cleared up completely, his doctor gave him streptomycin at weekly intervals.

Dr. Keller: There was a large infiltrate in the left upper lobe. The right lung appeared completely clear, as did the lower portion of the left. I didn't see any cavitation in that lesion. From a radiographic standpoint, I think it could have been either just an inflammatory disease or one of tuberculous origin. A lateral film showed that most of the infiltrate was in the posterior portion of the upper lobe.

A film taken approximately a month later showed a considerable resolution of the infiltrate in the left upper lobe, but there was some residual. Another film taken approximately two months after that second one displayed no appreciable change in the radiographic appearance. There may have been a small cavity indicated on that film, but I couldn't be certain. A lateral taken at the same time again showed an extensive infiltrate.

From a film taken just prior to the patient's admission here, we could see that the infiltrate had again spread extensively to that portion of the lung. The lung showed much more extensive disease than had been indicated on the previous examination, and we again wondered whether there might not be a cavity in the lesion.

Dr. Bean: Your interpretation, Dr. Keller, is that it was much more likely to have been an inflammatory thing, possibly tuberculous, than to have been a bronchogenic carcinoma.

The patient's course from there on was one of continuing fever, increasing cough, mental confusion and night sweats. He had one period of coughing up a great deal of greenish sputum. He was given many antibiotics, but he didn't improve. He was admitted to this hospital on April 22. He had many symptoms suggesting difficulty with some of his higher cerebral functions. Speaking, silent reading and vision were all variously, but not specifically, impaired so that he had diminution but not a complete loss of function.

The man arrived on our doorstep perilously close to death. He was in coma. He was dehydrated. He had a temperature of 102°F. He was breathing 52 times per minute. His pupils were of different sizes, and they didn't react to light. This finding might suggest that he had an ancient lesion of the iris—possible with tuberculosis, syphilis or sarcoidosis. We have no clue suggestive of syphilis. The unreactivity and the discrepancy in size of the iris on the two sides probably had resulted from a lesion in the brain. The right tympanic membrane was scarred, but the left appeared normal. Possibly he had an old middle-ear infection spreading to the petrous tip, and he might have developed meningitis.

There was an almost completely solid rigidity of the neck. It could not be flexed forward. The lung expansion was fair, dullness was present over the left upper lobe, and the breath sounds were diminished quite as one might have expected, since as Dr. Keller has told us, the infiltrate had extended and had come to occupy practically the entire left upper lobe, having at first been largely in the posterior portions of the left upper lobe. Rhonchi were heard over the right upper lobe.

The blood pressure was 200/80 mm. Hg—a very peculiar set of readings indicating systolic but not diastolic hypertension. Commonly, one sees it in people who have rigid large vessels. In elderly people with a great deal of aortic arteriosclerosis, one usually finds a high systolic and a relatively normal diastolic pressure, but in a 42-year-old man there is no reason to anticipate any such thing. One wonders whether somehow the process in the patient's lung might have acted as a plastic sclerosing tourniquet, encasing the upper portion of the aorta. That last is a fancy guess.

Despite the fact that the patient was breathing very rapidly and with great difficulty, the heart rate was not fast. The rhythm was regular. The heart wasn't enlarged. It was not thought to be failing. The abdomen was unremarkable.

The tests for meningeal irritation were positive, but the patient didn't have signs of pyramidal tract involvement. As a further sign of disorganization of his cerebral function, he had Cheyne-Stokes respiration. This state can be caused by any of a great variety of things—many lesions of the central nervous system, and particularly of the brain stem. It is notoriously common in people who have had large doses of morphine, or even small doses if they are sensitive. It may occur in primary pulmonary or primary cardiac failure. Thus, Cheyne-Stokes respiration is another indication of widespread difficulty within the brain, though it helps us little in localizing the trouble.

The hemoglobin was normal, but the white count was conspicuously elevated. Now we come to the problem of trying to decide whether to attempt showing that all of the findings were consistent with tuberculosis alone, or showing that he had tuberculosis and something else, or showing that he had something else and did *not* have tuberculosis. Dr. Ian Smith tells me that one occasionally gets a leukemoid reaction in tuberculosis when it is actively progressing and advancing. A white count of 34,000 ordinarily would suggest a septic or a pyogenic process, and *not* tuberculosis. There was a significant shift to the left, and it is consistent, too, though it does favor a pyogenic process.

The patient had an elevated spinal fluid pressure, with an enormous outpouring of cells. Either cells or bacteria or both had used up the sugar in the spinal fluid. He had conspicuous elevation of the protein in his spinal fluid, a circumstance suggesting that his disease probably had been going on for some time. The spinal fluid was cloudy, and a little skin or scum formed upon it after it had been allowed to stand. This finding is in favor of tuberculosis. The spinal fluid smear revealed no tubercle bacilli. A gram stain showed many gram-positive cocci in chains. We are confronted with the question of whether this man had a tuberculoma in a fairly large part of his brain, spreading into the meninges, after the manner described originally by Arnold Rich, leading to tuberculous meningitis. He had septic meningitis, with or without tuberculosis.

The laboratory studies were singularly unrevealing, with the exception of a slight elevation of the blood sugar. The therapeutic armamentarium was wheeled out, and all of the heavy guns were trained upon this patient's disease, but he died after 12 hours. I think his death was already certain by the time he got here, and I doubt that anything we did either hastened it or retarded it.

Before hearing Dr. Keller's report of the x-ray findings, I thought we needed to consider several things. One was a primary bronchogenic carcinoma with a spread to the brain ultimately settling in a lesion which had become necrotic, another was a pyogenic infection with streptococcal organisms. An abscess bursting and gaining access to the subarachnoid space might have produced a meningitis, killing him fairly promptly.

On the basis of the films, it seems very unlikely that he had primary bronchogenic carcinoma. At one stage, he had a fairly clear-looking lung, but later the density increased in size and depth. However, x-ray findings in bronchogenic carcinoma are notoriously unpredictable. Perhaps he had pulmonary tuberculosis, inadequately treated, with spread to the brain—a tuberculoma, ultimately with softening. Terminal infection with a streptococcus caused brain abscess and, ultimately, terminal meningitis.

One should mention a few other things. A fungus infection conceivably might have been the initial difficulty. The patient's course was unlike what I have seen in histoplasmosis or actinomycosis, but there are yeasts and fungi which produce a disease the natural history of which remains pretty thoroughly unwritten. One must think of this possibility in an obscure granulomatous disease. There is no need to bring coccidioidomycosis or blastomycosis into this discussion.

Brain abscess secondary to a non-acid-fast lung abscess is something else that must be taken into consideration. In these days of many antibiotics, we must think of brain abscess from a stunned but not killed microbe as a possible cause even of a chronic illness like this patient's. Fortunately fairly rarely, a patient will present to a neurosurgeon with signs of an expanding lesion. An operation will be required, especially if nothing else of any consequence is found. At surgery, an abscess may be found. Sometimes there is a primary abscess in the lung, too. Primary bronchogenic carcinoma, casting a small x-ray shadow or none at all, may be the original lesion.

I should like to conclude with a parody on Hamlet's soliloquy. As you remember, Hamlet says:

"To be, or not to be: that is the question:
Whether 'tis nobler in the mind to suffer
The slings and arrows of outrageous fortune,
Or to take arms against a sea of troubles,
And by opposing end them. To die, . . .
To sleep, perchance to dream; ay there's the rub";

Here is my parody:

TB or not TB?—that is the question.
Whether by tour de force I can arrive
At the right answer, I am most unsure.
What's more, the CPC may well disguise
In shoals of herrings red, some booby traps
To trip the unwary doctor as he stalks,
Head in the clouds, not reading right the signs.
And so with mirth and merriment for all
He'll wander to his own destruction.

TB or not TB?—that is the question.
Whether 'tis acid-fast or something else
That taking mastery of this poor man
Did by consuming kill him, he died—is dead.
What more? Of his own life we have a sketch,
The protocol, the thousand buffetings
His flesh was heir to. Was it the tubercle?
I might suppose it so
Though as impediments to this idea
No spitting blood, no friction rub.
Forsooth, we have tough diagnosis here.

When we have fumbled all the easy clues,
It stops us cold. There's the defect
That makes a torment of the speaker's life.
For who can learn the latest devilish lore
Of vectors long, of prideful EKG's,
X-rays, the sterile cultures and the rest;
The pangs of subtle pain, reports delayed,
The aberrations of electrolytes,
Reflexes gone awry, the neck all stiff
Despite the latest of the "mycin" clan
Purported to eradicate each germ
That plagues our lives? Who knows the new
Deflections of the hormone's path, or the
Cortex of the adrenal gland? How now?

Was it tumor in the lung and brain,
A vagrant fungus, or a wandering yeast?
Or was the streptococcus what he had?
No hurrying answer comes from Porter's lab,

Or lambent flames from the photometer,
To tell us where we are and why he died.

We wait for the pathologist to come,
Instructing us in his dark mysteries,
As dead men teach the living ones of death,
And some of life's obscurities unfold.

Dr. Bedell: Dr. Bean has demonstrated that there are hazards for him who writes the protocol as well as for him who discusses it. In the future, I'll try to keep my feet out of the spinal fluid.

Dr. George R. Zimmerman, Pathology: The pertinent abnormal findings were in the chest and in the brain. The prosector, on opening the chest, found that there were very dense fibrous adhesions between the upper lobe of the left lung and the thoracic cage. Upon removing the lung, he found that the upper lobe parenchyma was fibrotic. There were some eight to 10 abscesses varying in diameter from one to three centimeters. In general, these abscesses had rather thin, fibrous walls and roughened linings. In some instances, they were filled with serosanguineous fluid, but others were apparently connected with the bronchi and were empty. The lower lobes of both lungs were congested, and on section there were some firm areas that were more grey and granular in appearance. In other words, there was an acute pneumonitis, probably of some 12 to 24 hours' duration. The hilar lymph nodes were enlarged and soft, a finding that simply reflects the acute inflammation in the lungs.

The leptomeninges were thin and translucent. The right frontal lobe was swollen, and in the sulci over both cerebral hemispheres there were small collections of yellowish purulent exudate. Around the base of the brain, there was a large accumulation of yellowish-green exudate. Upon slight pressure on the right frontal lobe, pus could be made to ooze from a rent or sinus in the cortex near the midline. Presumably, this was the source of the meningitis. There was another abscess on the left side in the occipital lobe, similar in size to the frontal-lobe abscess—i.e., two to three centimeters in average diameter. The occipital lobe abscess communicated with the posterior horn of the left lateral ventricle, and there was some pus in the rest of the ventricular system.

The lungs and lung abscesses were cultured, and weakly hemolytic alpha Streptococci were recovered. They were the only organisms found. Cultures for fungi were negative. Cultures and guinea pig inoculation were negative for tuberculosis. Brain abscesses yielded alpha hemolytic Streptococci. They were negative for fungi, and there was a negative acid-fast smear of the cerebrospinal fluid. No other organism could be found by applying various stains for bacteria and fungi to the tissue sections.

Sensitivity studies were done on the organism that had been cultured, and it was found to be sensitive to penicillin, to tetracyclines and to chloramphenicol in ordinary concentrations. It was relatively insensitive to streptomycin, for its growth was impaired only by high concentrations of the drug.

The spleen and the heart blood yielded no growth, which means either that there was no bacteremia, or more likely, that massive doses of antibiotics were given shortly before the patient's death.

The gross photographs of the brain demonstrated the meningitis and the brain abscesses already described. The photomicrographs were selected to show the age and character of the infectious process. The walls of the lung abscess were heavily collagenized, and collagenization is a process that requires many weeks for completion. The infection in the parenchyma around the abscesses in the upper lobe of the left lung also was of long duration and had resulted in an organizing pneumonia, i.e., the alveoli were partly or completely filled with masses of chronic granulation tissue and fibrous tissue, and there was also fibrous thickening of some of the alveolar walls. The inflammatory exudate lining the lung-abscess cavity contained chains of gram-positive cocci.

The walls of the brain abscesses also showed dense fibrosis and gliosis indicative of a duration of many weeks. In addition, in parts of the brain abscess wall, there were recent necrosis and acute cellulitis without any fibrosis or other evidence of effective repair processes. Chains of gram-positive cocci were plentiful in the brain abscess exudates.

The meningitis was relatively acute, and the presence of a few mononuclear phagocytes and a few plasma cells and lymphocytes correlates nicely with the history by suggesting that the infection was of several days' duration.

In summary, then, this man started out with a bronchopneumonia—sometime in November, 1960, I believe—and then developed a chronic pneumonia with lung abscesses, presumably all due to streptococci, though we have no proof. Next, he developed metastatic brain abscesses and meningitis, and terminally the pneumonia spread to the lower lobes of both lungs.

Dr. Bean: I'd like to mention two things. First, this man illustrates what I have been talking about rather frequently—iatrogenic complications of one of Nature's own diseases. I suspect that inadequate treatment of his initial pneumonitis allowed it to become chronic and ultimately to produce brain abscesses. Second, since I didn't suggest streptococcal abscess and meningitis as my first diagnosis, I conclude with the charming comment I heard Louis Hamman make when, as happened only rarely, this sort of rebuff had been given him by Arnold Rich: "Why, you know, Dr. Rich, this just goes to show that sometimes the *best* diagnosis is not the *right* diagnosis."

Coming Meetings

In State

Nov. 8-9 — **Institute on Intrapartum and Neonatal Complications (S.U.I. Departments of Obstetrics and Gynecology, Pediatrics and Nursing; State Department of Health, Division of Maternal and Child Health).** Medical Postgraduate Conference. University Hospitals, Iowa City

Nov. 9 — **Medical Conference (Webster County Medical Society).** Warden Hotel, Fort Dodge

Nov. 15 — **AAGP meeting with Woodbury County Medical Society.** Sioux City

Nov. 17-18 — **Current Concepts in the Diagnosis and Treatment of Cardiovascular Diseases (Iowa Heart Association and the S.U.I. Department of Internal Medicine).** Medical Postgraduate Conference. University Hospitals, Iowa City

Nov. 19 — **Myasthenia Gravis.** Auditorium, Frankin Junior High School, Des Moines, 2:30 p.m.

Dec. 1 — **Respiratory Diseases (Iowa Thoracic Society and S.U.I. Department of Internal Medicine).** University Hospitals, Iowa City

Dec. 5-6 — **Surgery (Iowa Div. Am. Cancer Soc., and S.U.I. Department of Surgery).** University Hospitals, Iowa City

Out of State

Nov. 1 — **Lederle Symposium.** Colony Motor Hotel, Providence, Rhode Island

Nov. 1-2 — **Fifteenth Annual Postgraduate Assembly.** San Diego County General Hospital, San Diego, California

Nov. 1-4 — **American Society for Tropical Medicine and Hygiene.** Willard Hotel, Washington, D. C.

Nov. 2-4 — **Diagnosis in Ophthalmology.** University of California, San Francisco

Nov. 3-4 — **Central Society for Clinical Research.** Drake Hotel, Chicago

Nov. 4 — **Problems of Adolescence.** Children's Hospital, University of California, San Francisco

Nov. 4 — **Society for the Scientific Study of Sex.** Barbizon Plaza, New York City

Nov. 4-5 — **Twelfth County Medical Societies Conference on Disaster Medical Care (Council on National Security and American Medical Association).** Palmer House, Chicago

Nov. 5-8 — **Association of Military Surgeons of the United States.** Mayflower Hotel, Washington, D. C.

Nov. 6-8 — **Gynecology (University of Kansas School of Medicine).** Battenfeld Auditorium, University of Kansas Medical Center, Kansas City, Kansas

Nov. 6-8 — **American Occupational Therapy Association.** Sheraton-Cadillac Hotel, Detroit

Nov. 6-9 — **EENT Problems (Section on Ophthalmology and Otolaryngology of the Southern Medical Association).** Dallas

Nov. 6-10 — **The Internist's Role in the Pre and Postoperative Care of the Surgical Patient (American College of Physicians).** Mayo Clinic, Rochester, Minnesota

Nov. 6-10 — **American Association of Inhalation Therapists.** Statler-Hilton, Buffalo, New York

Nov. 6-10 — **Urologic Radiology for Radiologists.** Center for Continuation Study, University of Minnesota, Minneapolis

Nov. 6-11 — **Electrocardiography.** New York University Medical Center, New York City

Nov. 6-17 — **Surgical Technic.** Cook County Graduate School of Medicine, Chicago

Nov. 6-17 — **Gynecology, Office and Operative.** Cook County Graduate School of Medicine, Chicago

Nov. 6-17 — **Surgical Board Review, Part I.** Cook County Graduate School of Medicine, Chicago

Nov. 6-17 — **Practical Cystoscopy.** Cook County Graduate School of Medicine, Chicago

Nov. 8-10 — **Association of State and Territorial Health Officers.** Washington, D. C.

Nov. 9 — **Lederle Symposium.** Penn Harris Hotel, Harrisburg, Pennsylvania

Nov. 9-10 — **Theory and Practice of Contact Lenses.** New York University Medical Center, New York City

Nov. 9-10 — **Review of Recent and Practical Problems in Medicine (University of Southern California).** Statler Hotel, Los Angeles

Nov. 9-11 — **Symposium on Vascular Surgery.** New York University Medical Center, New York City

Nov. 9-11 — **American Academy for Cerebral Palsy.** Chase and Park-Plaza Hotels, St. Louis

Nov. 10 — **Fifth Annual Symposium on Diabetes (Chicago Diabetes Association).** Offield Auditorium, Passavant Hospital, 303 East Superior Street, Chicago

Nov. 10-12 — **Gerontological Society, Inc.** Pittsburgh

Nov. 11 — **Postgraduate Conference on Allergy.** Presbyterian Medical Center, San Francisco

Nov. 11-13 — **Alcohol and Civilization.** University of California, San Francisco

Nov. 12 — **Lederle Symposium.** Robert Meyer Hotel, Jacksonville, Florida

Nov. 13-16 — **Interstate Postgraduate Medical Association of North America.** Cleveland Auditorium, Cleveland

Nov. 13-16 — **Internal Medicine (University of Kansas School of Medicine).** Battenfeld Auditorium, University of Kansas Medical Center, Kansas City, Kansas

Nov. 13-17 — **American Association of Public Health Physicians.** Cobo Hall, Detroit

Nov. 13-17 — **Recent Advances in the Diagnosis and Treatment of Heart and Lung Diseases (American College of Chest Physicians).** Park Sheraton Hotel, New York City

Nov. 13-17 — **Blood Vessel Surgery.** Cook County Graduate School of Medicine, Chicago

Nov. 13-17 — **Surgery of the Cornea.** New York University Medical Center, New York City

Nov. 14-16 — **Fractures in General Practice.** Medical College of Georgia, Augusta

Nov. 14-17 — **Surgical Rehabilitation of Arthritic Deformities.** New York University Medical Center, New York City

Nov. 15 — **Lederle Symposium.** American Hotel, Allentown, Pennsylvania

Nov. 15-16 — **American College of Preventive Medicine.** Detroit

Nov. 15-17 — **Ophthalmology (Refraction) for General Physicians.** Center for Continuation Study, University of Minnesota, Minneapolis

Nov. 16 — **Treatment of Mental Retardation by the General Practitioner.** Auditorium, Nebraska Psychiatric Institute, Omaha

Nov. 16 — **Lederle Symposium.** Far Hills Inn, Somerville, New Jersey

Nov. 16-18 — **Symposium on Myocardial Infarction.** Detroit

Nov. 16-18 — **Orthopedics for Orthopedic Surgeons and General Physicians (Hand Surgery).** Center for Continuation Study, University of Minnesota, Minneapolis

Nov. 16-18 — **Milwaukee Divisional Meeting, American Psychiatric Association.** Hotel Schroeder, Milwaukee

Nov. 17-21 — **National Society for Crippled Children and Adults.** Denver-Hilton Hotel, Denver

Nov. 18-19 — **Psychiatry in General Practice (University of California, San Francisco).** Napa State Hospital

Nov. 21 — **Hearing Problems (The Northwest Missouri Chapter of the Missouri Academy of General Practice and the University of Kansas School of Medicine).** The Moila Temple, St. Joseph, Missouri

Nov. 24 — **Symposium on Anticoagulant Therapy.** University of Southern California, Los Angeles

Nov. 25-27 — **American College of Chest Physicians.** Annual interim session. Brown Palace Hotel, Denver

Nov. 26-Dec. 1 — **Radiological Society of North America, Inc.** Palmer House, Chicago

(Continued on page li)

THANKS

With the advent of the Thanksgiving season, it is appropriate for us to express our gratitude to those who have served our profession so well.

To those physicians who have contributed generously of their time, energy and zealous effort on behalf of organized medicine at the county, state and national levels, we are deeply indebted. For the most part, theirs is a thankless task, devoid of praise, not without criticism, and sometimes bringing upon them some abuse. Somehow, their unselfish attempts to render service has been given the unfortunate appellation "medical politics," though as a matter of fact the only reward they can hope to achieve is the satisfaction which comes from a job well done. The JOURNAL takes this opportunity to thank and to praise those physicians. Without them and their dedicated efforts, the public would suffer and the profession would be in a chaotic state.

For their loyal support and conscientious effort, the lay staff of the Iowa Medical Society deserve great credit. We owe very much to them, and we offer them our profound thanks.

This is also a fitting time to express appreciation and gratitude to Dean Nelson and the faculty of the SUI College of Medicine for their loyal support and generous scientific contributions to the JOURNAL.

A WORD TO THE WISE

Since the advent of the antibiotic drugs, physicians have been under constant pressure to call upon leger-de-main in curing all the ills to which flesh is heir. The temptation to the newest or most loudly heralded drug is very great. The efficacy and safety of each new drug should have been thoroughly demonstrated before the practicing physician employs it in the treatment of his patient. The enthusiasm of the detail man, as he tells the merits of a new medication, is insufficient evidence of its value or safety. The physician must study the literature and be guided by the experiences of others.

Kauffman* wisely admonishes: "Treatment with some potent drugs may put the patient's emotional stability to a severe practical test despite the doctor's best efforts to prepare him for the rigors of therapy. Not all adverse reactions are predictable, but at least some result from inadequate clinical study of the patient before therapy with drugs. A doctor's prestige, professional reputation and even his livelihood may depend on his skillful use of medications in the treatment of sick patients. The wise doctor is not stampeded into getting on the therapeutic merry-go-round, but thoughtfully selects those medications which seem likely to offer his patients the greatest benefits with the fewest risks."

* Kauffman, W.: Some psychological aspects of therapy with drugs, part iv. CONNECTICUT MED., 25:505-509, (Aug.) 1961.

MYASTHENIA GRAVIS

The dramatic role of the element potassium and its importance in the various phases of human physiology have been repeatedly demonstrated. In THE LANCET, Gottlieb and Laurent* have recalled that in 1934 British physiologists demonstrated that the addition of potassium chloride to a perfusing solution stimulated the cervical ganglion of the cat, and that smaller amounts of the chemical raised the response of ganglionic cells to preganglionic stimuli, to acetylcholine and other stimulants. These studies prompted a clinical trial of potassium chloride in the treatment of myasthenia gravis in the hope than an action might result at the myoneural junction similar to that which had occurred at the synapses of the cervical ganglia. Large doses of the drug by mouth did result in a demonstrable improvement, and small repeated doses appeared to be a useful adjunct to therapy with neostigmine.

The rapid excretion of potassium and the unpleasant side effects that occurred when it had been given in large doses prompted Gottlieb and Laurent to try spironolactone, an aldosterone antagonist, in the treatment of a patient with severe myasthenia gravis, because the drug diminishes the excretion of potassium. A bedridden patient who had difficulty in talking and swallowing and who required injections of neostigmine as well as neostigmine by mouth, was enabled to walk, to move her arms more easily, and to talk and swallow more satisfactorily after three days' administration of spironolactone. The drug appeared to potentiate the action of neostigmine. Cessation of the drug brought on a relapse.

These investigators treated six additional patients suffering from myasthenia gravis with the aldosterone antagonist, and observed definite improvement in five of them. The one patient proving refractory to treatment was in marked remission. From their experience, they concluded that

* Gottlieb, B., and Laurent, L. P. E.: Preliminary communication: spironolactone in treatment of myasthenia gravis. LANCET, 2:528-529, (Sept. 2) 1961.

100 mg. of spironolactone four times daily resulted in improvement, and a double-blind trial in two patients supported that observation. Investigations of potassium excretion and measurements of the potassium content in the muscles of patients with and without spironolactone therapy are to be carried out. The authors suggest that as a result of their experience the drug deserves wider use in the treatment of myasthenia gravis.

A PUBLIC RELATIONS OPPORTUNITY FOR IOWA PHYSICIANS

Between 30 and 35 representatives of organizations greatly interested in helping farm and small-town people maintain their health met at the Savery Hotel, in Des Moines, on October 11 to plan the Regional Rural Health Conference that will be held there on May 18-19, 1962. The AMA Council on Rural Health, the sponsoring group, was represented by four of its physician members, one of whom is Dr. Samuel P. Leinbach, of Belmond, and by one of its staff people. The Iowa Medical Society had a half-dozen members of its Committee on Rural Health in attendance, and there were delegations there from the agricultural extension service, the public health departments and various other farm or health groups and medical societies in Iowa, Minnesota, South Dakota, Wisconsin, Illinois, Missouri and Kansas. The meeting in May of next year is expected to draw delegations and individual citizens from a total of 13 midwestern states.

The subjects chosen for presentation at the Conference are ones that it was felt would be of greatest interest to the laymen and women who will be greatly in the majority at that meeting. The first session of the day-and-a-half program will start with a keynote address designed to project the theme of the gathering—that rural health problems in the sixties include many traditional ones, some of them more serious today than they have ever been, together with many new difficulties. Thereafter, there will be lectures on the controlling of health-care costs—eliminating the abuses of health insurance, providing medical care for the aged, getting a better distribution of doctors, and recruiting young people for the various health careers. Early on Friday afternoon there is to be a presentation on quackery and cults. Then will come a series of discussions of rural health hazards—poisons, accidents, the influx of migrant workers, the urbanization of rural areas, the general failure to keep immunizations current, and zoonotic diseases. A banquet will conclude the Friday program. The Saturday morning session will be given over to the health problems of civil defense. When one realizes that in the event of a nuclear attack, each family group will be altogether isolated and completely unable to get professional medical care, he begins to see the enormity of the difficulties that must be prepared for.

Now that the program-planning is well under way, it is time for individual physicians—particularly in Iowa, the host state—to take steps toward making next spring's Regional Rural Health Conference a successful undertaking for the AMA and the Iowa Medical Society. First and foremost, the legislative contact men should notify the legislators and the Iowa delegation in Congress about the dates—May 18 and 19—and the place—Hotel Savery, Des Moines. Furthermore, arrangements should be made to provide a physician-host for each of the lawmakers who can plan to attend.

Every physician should help to publicize the event, and local Auxiliaries should see to it that posters announcing it are displayed at each of the civic gatherings in their communities.

This Conference will be an occasion on which medicine can achieve a great deal as regards improving the public image of the doctor. Let's start early and work hard at making it an outstanding success! At AMA expense, outstanding speakers will be brought to Des Moines to discuss medicine's rural and small-town problems in laymen's terms, but it isn't enough that medicine is arranging the program and will pay the bills. Doctors must be present in large numbers! Only if many physicians are in evidence will the farm people and the members of ancillary health professions feel that medicine attaches great importance to the subjects that are discussed and really wants their cooperation in improving conditions.

Circle May 18-19 on your calendar, and begin talking to your patients, your lawmakers and your other friends about attending the AMA Regional Rural Health Conference that is to be held in Des Moines on those dates.

"In-laws are like seeds. You don't need them, but they come with the tomatoes."

—ST. LOUIS POST-DISPATCH

LECTURE ON MYASTHENIA GRAVIS

The Iowa Chapter of the Myasthenia Gravis Foundation has invited physicians from throughout the state to hear a lecture by J. E. Tether, M.D., F.A.C.P., perhaps the foremost authority on the disease, at the auditorium of Franklin Junior High School, Franklin Avenue and Forty-eighth Street, Des Moines, on November 19 at 2:30 p.m.

Dr. Tether is an assistant professor of neurology at the Indiana University Medical Center, and specializes in the treatment of myasthenia gravis. He will show films and slides to illustrate his talk.

The medical advisory board of the Iowa Chapter consists of Drs. Olin A. Elliott, Walter D. Abbott, Joel D. Teigland, J. W. Chambers, Arnold Smythe and T. B. Summers, all of Des Moines.

BIOCHEMISTRY OF THE BRAIN

Until 10 or 15 years ago, physiological-chemical studies of the brain were non-existent. Today we are still in the Dark Ages, but we at least can see an occasional ray of light filtering through the night. Thus, we have come to realize that all mental aberrations are not due to sexual imbalance or even to conflicts or frustrations. The discovery of the phenothiazines (the so-called "tranquilizers") and of the monoamino oxidase inhibitors (the so-called "mood elevators") have led us to recognize that profound changes in brain physiology may occur as a result of chemical manipulation alone. A bewildering array of substances have been placed on the market within the past four or five years, designed to alter the affects of mentally-disturbed patients. These substances have been a great boon to the psychotherapist and have lessened the necessity for hospitalizing patients in mental institutions for electric-shock therapy.

It is equally interesting, however, for the pure scientist to witness the hoped-for effects and the side-effects of these drugs. He asks himself questions: Why are some phenothiazines (e.g., Pacatal) prescribed for Parkinsonism? Why do some drugs such as the Rauwolfia products and some other phenothiazines produce extra-pyramidal reactions indistinguishable from Parkinsonism?

One theory designed to explain the occurrence of Parkinsonism following the administration of these drugs is "based on the postulate that normal functioning of the extra-pyramidal system depends upon an appropriate balance between two competing groups of neurohumoral agents which may be neuro-transmitters, one group being acetylcholine-histamine, and the other catecholamines and serotonin. It is suggested that drugs which produce extra-pyramidal reactions do so because they interfere with the catecholamine-serotonin system."* Accordingly, in patients who have developed a Parkinson-like syndrome as a result of the administration of tranquilizers, antidotes have been given designed either to enhance the catecholamine-containing system or to block the histamine-containing system. The latter approach, through the administration of diphenhydramine, has proved the more effective in controlling the extra-pyramidal reaction.

Tremendous strides are being made in the chemical analysis of brain function in health and disease. Perhaps some conditions such as obesity, depression and Parkinsonism may be due, in part, to an altered chemical metabolism. Perhaps some day we shall find some chemical answers for these problems. At least it seems that we are hearing the rumblings of a Renaissance.

* McGreer, P. L., Boulding, J. E., Gibson, W. D., and Foulkes, R. D.: Drug-induced extrapyramidal reactions. J.A.M.A., **177**:665-670, (Sept. 9) 1961.

AN ADENOVIRUS MAY CAUSE INTUSSUSCEPTION

In his textbook THE SURGERY OF INFANCY AND CHILDHOOD, Gross* states that the etiological factor of intussusception is still unknown in 90 to 95 per cent of childhood cases. But a recent communication by Gardner, a British virologist, suggests that an adenovirus may possibly play a role in its causation.

Gardner,** knowing that adenoviruses may be excreted in large quantities in the stool and for long periods of time, assumed that with present methods of isolation it was unlikely that the adenovirus was swallowed from the upper respiratory tract. On this premise it then appeared that the adenoviruses must multiply somewhere in the intestinal tract. With this concept in mind, the stools of all confirmed cases of intussusception in children were examined during a period of about one year.

Feces from 10 cases of intussusception were cultured on HeLa cells. Control studies were made by similar cultures on 13 patients ranging from three months to one year of age who had been admitted for the treatment of traumatic injuries or for the treatment of hernia. Of the 10 cases of intussusception examined, no virus was cultured from the stools of three patients; Herpes Simplex virus was found in one; and adenovirus was demonstrated in the stools of six. Four of the adenoviruses were type 5; one was type 4; and one was type 7. None of the 13 control patients had any viruses in their stools, when specimens were cultured in the same way as those from the infants with intussusception had been. In an addendum to his communication, Gardner reported that cultures were taken on an additional three patients with intussusception, and that adenoviruses were found in two of the three.

He suggests that possibly the adenoviruses multiply in the lymphoid tissue of the bowel—perhaps in Peyer patches and the terminal ileum, resulting in an enlargement of the tissue. This factor, either alone or in association with some other physiological disturbance, may initiate the intussusception. Abdominal symptoms are frequent in adenovirus infections, and the virus has been demonstrated in the mesenteric glands. It is postulated that perhaps intussusception is just one of the rare manifestations of adenovirus infections of the bowel in the young. Many more children in this group may manifest the infection by diarrhea, and older children may manifest it by mesenteric adenitis.

Whether or not the adenovirus can be confirmed as playing a significant role in the etiology of in-

* Gross, Robert E.: THE SURGERY OF INFANCY AND CHILDHOOD, Philadelphia, W. B. Saunders Company, 1953.

** Gardner, P. S.: Preliminary communication: adenovirus and intussusception. BRIT. M. J., **2**:495-496, (Aug. 19) 1961.

tussusception, or as a causative factor in mesenteric adenitis, offers an attractive field for study and, just possibly, an explanation for a common and serious problem.

PROMPT DIAGNOSIS OF STAPHYLOCOCCAL INFECTIONS

An editorial by Robert I. Wise, in a recent issue of THE ANNALS OF INTERNAL MEDICINE,* emphasizes the necessity for early, accurate diagnosis of serious staphylococcal infection, if prompt, effective therapy is to be initiated before the disease reaches the stage of bacteremia and metastatic abscesses. The infection can be transformed in a few hours from one that is amenable to rapid and effective treatment to a condition that is highly resistant to therapy and often life-threatening.

A Gram stain of properly collected material, which is representative of the infected site, is a valuable aid in rapid diagnosis, but it is too seldom considered and its merit is insufficiently appreciated. This method can be used when the patient is first seen by the physician, and thus offers a definite time advantage over the culture, which should be taken at the same time. The identification by Gram stain, the number of organisms seen, the characteristic grape-like clusters in association with polymorphonuclear leukocytes, and the evidence of phagocytosis can make one confident of the bacteriologic diagnosis of staphylococcal disease. The direct stain is particularly valuable in staphylococcal pneumonia, empyema, meningitis and enterocolitis. Valuable information is thus obtained within 10 or 20 minutes after the specimen has been obtained.

Cultures taken at the same time as the smear for Gram staining provide confirmation of the diganosis and permit isolation of the culture for a determination of antibiotic sensitivities. Ordinarily, the results of *in vitro* tests for susceptibility are not available until the third day.

Hemolytic coagulase-positive staphylococci are often found on the skin, in the upper respiratory tract and in the colon, in patients with febrile illnesses that are due to some other organism. Routine bacteriologic cultures may contain the staphylococci as a contaminant, and give rise to an erroneous diagnosis. The mere presence of the organism in culture thus doesn't constitute proof of its etiologic significance, and even if coagulase positive, one can't be certain that the staphylococcus is the infective agent. However, if the initial Gram stain has indicated that the infecting organism is a staphylococcus, a growth of hemolytic, coagulase positive staphylococci on culture is a welcome corroboration of the initial diagnosis.

The successful treatment of patients with severe staphylococcal infections in which surgical drainage cannot be instituted, demands the introduction of an antibiotic at the site of the infection promptly and in sufficient concentration to be bactericidal. Bacteriostasis alone is insufficient. One must remember that an antibiotic which inhibits the growth of Staphylococcus aureus in tests utilizing the commonly employed disc method for determining susceptibility may not provide sufficient concentration at the site of the infection. In other words, the *in vitro* sensitivity test doesn't provide information regarding bactericidal effectiveness. The administration of a combination of antibiotics, both in bacteriostatic concentrations, is not so efficacious as the use of a single antibiotic in a concentration sufficient to be bactericidal.

At the present time, Vancomycin is the antibiotic of choice in the treatment of severe staphylococcal infections, and there have been no reports in the literature to indicate that the organism has become resistant to the drug. A regimen of intravenous administration of Vancomycin should be initiated promptly if the Gram stain of properly collected material is indicative of staphylococcal infection. A continuous slow infusion of the recommended dose throughout the 24-hour period is advisable, and since a sufficient concentration may not be achieved at the site of infection in this way, due to the fact that the rate of neutralization and excretion exceeds the rate of infusion, a booster dose once or twice a day may be necessary in addition to the continuous infusion. Some physicians prefer the intravenous injection of the drug in a small amount of fluid every four hours. In staphylococcal enterocolitis, the drug is administered by mouth, and it has proved to be very effective in the treatment of this condition.

Thrombosis of veins has been one of the difficulties in the administration of Vancomycin, but the problem may be reduced by the introduction of a catheter into an antecubital vein, or in critical cases in which therapy must be continued for a long period of time, the catheter has been introduced into the superior or inferior vena cava. Various toxic manifestations have been described but are ordinarily of insufficient seriousness to interrupt therapy. Transient rashes have occurred, nerve deafness has been reported, and a temporary rise in urea nitrogen has been recorded in one patient.

By the use of the Gram stain of properly collected material, a diagnosis of a staphylococcal infection can be made from 36 to 72 hours before the report of culture and sensitivities becomes available. In cases of severe staphylococcal infection, the prompt initiation of Vancomycin therapy in a sufficient concentration to be bactericidal offers great benefit to a critically ill patient and has brought about the recovery of many individuals with otherwise fatal disease.

* Wise, R. I.: Diagnostic dilemma of severe staphylococcal infections. ANN. INT. MED., 55:344-346, (Aug.) 1961.

PREVENTION OF POSTOPERATIVE WOUND INFECTION

The tendency for surgeons to depend upon antibiotics for the prevention of postoperative wound infection, rather than to rely upon sound surgical principles, is deprecated by Dykes and Anderson, in the CLEVELAND CLINIC QUARTERLY for July, 1961.* They have outlined the principles maintained by their department of plastic surgery, where only one infection occurred during a year when 1,248 operative procedures were performed. That one infection occurred in a patient who had undergone laryngectomy and a radical neck operation.

The factors which give rise to wound infection are a receptive host, contaminating organisms, and a wound that provides a suitable culture medium for the growth of bacteria. Modern preoperative preparation of the patient corrects the electrolyte imbalance, low blood volume, anemia and avitaminosis, thus minimizing the factor of host susceptibility in the development of wound infection.

Most wound infections have their origin in the operating room at the time of surgery. Even there, it is impossible to achieve asepsis and sterility, and all surgical wounds are contaminated. From the Cleveland experience, the authors have concluded that thorough gentle scrubbing, careful preparation of the operative site and vigilance against breaks in technic are vastly more important than emphasis upon reducing environmental contamination in the operating room, though the latter aspect cannot be disregarded. The absurdity of a surgeon's scrubbing for 10 minutes, wearing two masks, a sterile gown and gloves, and then operating upon a patient whose skin has been prepared for 30 seconds with a pink antiseptic is obvious. It is the practice of the authors to scrub the operative site thoroughly but gently for 10 minutes with an antiseptic soap, and then to apply an antiseptic solution. They emphasize that the scrubbing should begin in the center of the operative field and should be carried out in a centrifugal manner. By scrubbing back and forth, one can drag bacteria from the peripheral, contaminated field into the clean area.

The greatest single deterrent to wound infection is the creation of a healthy, viable wound produced by an atraumatic technic and closed by a meticulous approximation of all layers and by the obliteration of all potential spaces. It is only by such a technic that the amount of culture medium in the wound can be minimized so that the contaminating organisms have no opportunity to multiply. The gentle handling of tissues, the use of moist rather than dry sponges, the obliteration of any dead spaces, complete hemostasis with a minimum of ligatures, a properly applied pressure dressing—all of these are details of surgical technic that are of great importance and are too often carelessly disregarded.

If it is possible for a group of surgeons in a Cleveland hospital almost to eliminate postoperative wound infection through meticulous attention to detail and adherence to surgical principles, it is reasonable that similar practices and comparable results can be expected in other institutions at the hands of other surgeons. Frequent appraisals of procedures, technics and results are necessary if postoperative wound infection is to be prevented.

* Dykes, E. R., and Anderson, R.: Atraumatic technique—sine qua non of operative wound prophylaxis. CLEVELAND CLINIC QUARTERLY, 28:157-165, (July) 1961.

MULTIPLE SCLEROSIS

In a thorough review of the demyelinating diseases, Schumacher* summates the present concepts of the therapy of multiple sclerosis as follows: "Much benefit has come to the patient in the form of supportive and symptomatic treatment. To date, these ministrations of a kindly physician have probably been, of all aspects of treatment, the most helpful to sufferers from the disease, and remains the long-range foundation for assisting patients along their rough and uneven path through life. In general such patients are hopeful and respond unusually well to sympathetic interest, encouragement and minor signs of improvement."

He points out that it is inconceivable that the brilliant minds and powerful tools of science, which have already contributed so greatly to the knowledge of neurologic disease, won't clarify the cause and provide a solution for this serious disease in the reasonably near future. To date, physicians have proceeded along apparently logical or empirical lines in their endeavor to help patients with multiple sclerosis. Schumacher warns that until the specific cause or causes are elucidated or some laboratory criterion for effective therapy has been demonstrated, the application by an individual physician of any new and theoretically specific mode of therapy directed at the pathologic process itself is unjustified. Such approaches to the treatment of multiple sclerosis should be restricted to a thoroughly controlled, carefully planned investigation of a sufficient number of patients over a sufficient number of years.

* Schumacher, G. A.: Demyelinating diseases. New England J. Med., 262:1119-1126, (June 2) 1960.

"We are as young as our self-confidence, as old as our fear; as young as our desire, as old as our despair."

President's Page

The 1962 Annual Meeting of the Iowa Medical Society will be held on May 13, 14, 15 and 16, at the Veterans Memorial Auditorium, in Des Moines. The theme of the meeting will be "The Present Day and Future Practice of Medicine."

Outstanding speakers from around the country and two provinces of Canada will be on the program.

Plan to attend the whole scientific session.

Watch for future announcements in your IMS JOURNAL.

President

THE JOURNAL *Book Shelf*

BOOKS RECEIVED

REHABILITATION OF A CHILD'S EYES, THIRD EDITION, by *Herbert M. Katzin*, M.D., and *Geraldine Wilson*, R.N. (St. Louis, C. V. Mosby Co., 1961. $3.75).

APPRAISAL OF CURRENT CONCEPTS IN ANESTHESIOLOGY, ed. by *John Adriani*, M.D. (St. Louis, C. V. Mosby Co., 1961. $7.75).

MEDICAL PHYSIOLOGY, ELEVENTH EDITION, ed. by *Philip Bard*. (St. Louis, C. V. Mosby Company, 1961. $16.50).

THE PARENCHYMA OF LAW, by *David W. Louisell* and *Harold Williams*. (Rochester, New York, Professional Medical Publications, 1961. $).

WILLIAMS OBSTETRICS, TWELFTH EDITION, by *Nicholson J. Eastman*, M.D., and *Louis M. Hellman*, M.D. (New York, Appleton-Century-Crofts, Inc., 1961. $16.00).

PRACTICAL PEDIATRIC DERMATOLOGY, SECOND EDITION, by *Morris Leider*, M.D. (St. Louis, C. V. Mosby Co., 1961. $13.75).

PSYCHOPATHOLOGY OF AGING, ed. by *Paul H. Hoch*, M.D., and *Joseph Zubin*, Ph.D. (New York, Grune & Stratton, 1961. $9.75).

BOOK REVIEWS

PREVENTIVE MEDICINE IN WORLD WAR II, Volume V—Communicable Diseases Transmitted Through Contact or by Unknown Means, prepared and published under the direction of Lt. Gen. Leonard D. Heaton, surgeon general of the Army. (Washington, D. C., U. S. Government Printing Office, 1960. $5.75).

This volume, the first one devoted to communicable disease in the series collectively entitled "Preventive Medicine in World War II," covers diseases transmitted chiefly through the respiratory and alimentary tracts. The book is concerned with communicable diseases "transmitted through contact or by unknown means." Twenty-seven physicians who served in the armed forces medical service during World War II have presented much of the material in the volume. Although some of the diseases described, such as leprosy, schistosomiasis and yaws, are infrequently seen in this country, most of them regularly attack our civilian population. These diseases include hookworm, fungus infections, impetigo, scabies, leptospirosis, trachoma, poliomyelitis, Q fever, infectious mononucleosis, venereal diseases and viral hepatitis.

Edited by Ebbe Curtis Hoff, M.D., this book numbers among its contributors many eminent medical men who are recognized authorities in their respective specialties. Among them are Dr. Thomas B. Turner, dean of the medical faculty and professor of microbiology at Johns Hopkins University; Dr. Thomas G. Ward, professor of virology at the University of Notre Dame; and Dr. James A. Doull, medical director of Leonard Wood Memorial and formerly medical director of the U. S. Public Health Service.

Although the book contains excellent reference material for physicians, medical students, epidemiologists and venereal disease control officers, it also appeals to the entomologist and the parasitologist. In understanding an outbreak of a communicable disease in a civilian population, one frequently must resort to armed-service reports for the behavior of the disease under field conditions. The book has a very definite current value, and 50 to 75 years from now it will still have great value as a historical source.—*Ralph H. Heeren*, M.D.

THE CARDIAC ARRHYTHMIAS, by *Brendan Phibbs*, M.D. (St. Louis, The C. V. Mosby Company, 1961. $7.50).

Dr. Phibbs, formerly a part-time teacher at the Northwestern University School of Medicine and now a practitioner of cardiology in Casper, Wyoming, has had the concept that some practicing physicians need a little basic review of the cardiac arrhythmias. He has, therefore, written a monograph intended as a "Guide for the General Practitioner" which covers the basic electrocardiographic concepts of the arrhythmias. The monograph is simply and basically written, with large spaces left between the lines so that the reader may assimilate the material quickly. It is also liberally sprinkled with diagrams and electrocardiographic tracings illustrating the basic principles discussed. At the end, there are a group of electrocardiograms presented as "unknowns," so that the reader may test himself. A few paragraphs concerning "Clinical Recognition" and "Treatment" have been added following each subject discussion.

In this day and age, it seems that practicing physicians are either "extra-general practitioners" or "super-specialists." To "super-cardiologists," this book would be of no value, but to any of the other categories of practicing physicians, such as "generalist" or "super-specialist," this primer may be of extreme value as a general review. The dermatologist, the surgeon, the gastroenterologist, the anesthesiologist or the generalist undoubtedly has forgotten much of the basic material that this book contains, and there is no question that each of them will come across patients with one or another of the arrhythmias. Some of these are serious, some innocuous, but he should know which is which. This little book points out the methods of recognition that may mean life or death to the patient concerned. It is up to us to keep posted on the basic knowledge of a specialty which may not be our own.—*Daniel A. Glomset*, M.D.

THE GENESIS OF THE BRITISH NATIONAL HEALTH SERVICE, by *John* and *Sylvia Jewkes*. (Oxford, Basil Blackwell, 1961. $1.00).

The British National Health Service is still the subject of controversial writing, most of which relates to contingent problems. The reasons advanced by the founders of the Service, at the time the National Health Service Act was passed in 1946, are no longer the subject of study or discussion. Little or no attention, however, is given to fundamental questions such as, "Was the system altogether necessary?" "How much benefit have the British derived from the Service?" "How does this benefit compare with the benefit that other countries have derived from their own systems?" and "What could have been alternatives to the National Health Service?" Professor John Jewkes of Oxford University, in cooperation with his wife, Sylvia Jewkes, has just published a study in which the emphasis is mainly on these fundamental questions. This is the first in a proposed series of studies on the economics and administration of medical services. Its purpose, as suggested in the title, THE GENESIS OF THE BRITISH NATIONAL HEALTH SERVICE, is to trace the origin of the Service and to test, in the light of 12 years' experience, the validity of the arguments that swayed the British public toward support of the National Health Service Act.

The academic reputation of Professor Jewkes, his long experience in the field of medical economics, his knowledge of medical services in various countries on both sides of the Atlantic and, especially, the clear and well documented presentation of the material make this study, and let us hope the ones that will follow also, a must for anyone interested in the British experiment with medical services in the immediate post World War II period, an experiment which the authors describe as the most radical departure in the distribution of medical services that any country of the Western world has yet made.

John and Sylvia Jewkes test the validity of the arguments put forth by the founders of the Service by comparing medical services in England before and after World War II. A further comparison is made with the medical services available in the United States during the same time. Since in comparing two periods within the same country one must consider all changes in medical care that may have occurred in the intervening period, and since these changes are freely communicated from country to country, an international comparison if properly used may be more revealing of real progress than a comparison of two different periods within the same country. The rate of progress of medical services in England measured in terms of total expenditures, hospital facilities, number of doctors, nurses and dentists does not compare favorably with the rate of progress in the United States. The authors conclude that: "the average American now has more medical services than the average Briton and . . . the gap between the two (countries) has been widening."

Defenders of the British Health Service contend that the condition of medical services and medical resources available in England in 1939 justify the radical change brought about in the post-war period. Students of the National Health Service have pointed out, for instance, that since 1948 there has been a marked change in the distribution of medical services, implying that before the war England suffered from extensive maldistribution of its medical resources. The Jewkeses point out that the claim of maldistribution is usually dependent on an assumed "idea of optimum distribution." The practical realization of such an idea is dependent on too many variables.

Since the hospital situation in England has remained stationary, it is obvious that there has been no change in the distribution of medical services in terms of the location of hospital beds. Considering distribution from some other aspects—geographic, economic status of patients, availability of work per doctor—the Jewkeses conclude that since the establishment of the Service there has been "somewhat more uniformity," but in no case have the changes been spectacular. The specific findings for geographic distribution are: 1.) the distribution of dentists since 1948 has become more uneven; 2.) the distribution of doctors, as a whole, as indicated by the censuses of 1911, 1931 and 1951, shows "no great change"; 3.) the distribution of general practitioners has changed somewhat under the Service, the major changes occurring in areas with the lowest doctor-population ratio; and 4.) the distribution of specialists has changed markedly under the Service. The last of these findings has been abused by the defenders of the Service. The real change, the Jewkeses point out, has not been in the amount of specialist service available, but in the number of "titled" specialists. Under the rules of the Service, doctors who had previously engaged only in part-time-specialist work found it financially more attractive to be classified as specialists. Thus, at the inception of the Service the number of specialists on the government lists jumped considerably.

Turning to the administration of medical services before and after the inception of the NHS, the Jewkeses suggest that the alleged faults of the pre-War administration certainly did not warrant so drastic a change as was brought about by the 1946 Act. National Health Service officials have found that Aneurin Bevan's contention that "there is no fundamental difference between the National Health Service and the railways in terms of administration" is not so true after all. The "central pattern and purpose" that administrators have been attempting to impose on the Service for effective administration has created complications and problems.

Is the improved health status of the British people related solely to the change in distribution of medical services? The Jewkeses prefer to think that better economic conditions, housing, diet and overall sanitary conditions must also be taken into consideration on this score. What could have happened to medical services in England if there had been no National Health Service? Considering the trend followed in other countries—and here they rely mainly on the experience of Switzerland—the authors feel that it is most likely the National Health Insurance system established in 1911 primarily for the benefit of low income groups, could have been extended to cover not only the workers but also their dependents. Voluntary health insurance could have been extended, possibly with government aid, to cover a majority of the population. The need for more capital expenditure for hospitals and medical training facilities could have been met with government aid. Thus the Jewkeses conclude "it is reasonable to suppose that, even without a National Health Service, Britain would have enjoyed after 1948 medical services more ample

and better distributed than those which existed before the war."

A PRELUDE TO MEDICAL HISTORY, by *Félix Martí-Ibañez*, M.D. (New York, MD Publications, Inc., 1961. $5.75).

This books combines good literary style with very interesting historical material. The chronological account carries the spirit of medical progress from the dawn of man and the ancient civilizations up through the Middle Ages to the Atomic Age.

All physicians need periodic stimulation and inspiration, and one of the better ways to get these things is to read about the almost miraculous advance of medicine and the men who made it possible. The medical pioneers of each age come alive again in the pages of this book because of Dr. Martí-Ibañez' ability to describe them colorfully and to put them in their proper historical settings.

I heartily endorse this book for anyone who wants to review the history of medicine or for those who love history for itself.—*Edward R. Posner, Jr.*, M.D.

RELIEF OF SYMPTOMS, SECOND EDITION, by *Walter Modell*, M.D. (St. Louis, The C. V. Mosby Company, 1961. $11.50).

The second edition of this book is a revision of a practical guide for the task of relieving patients from distress. The author emphasizes the need for treating the patient as a whole, rather than his disease state alone. This is a refreshing approach to therapeutics, and one that is often overlooked.

This book should be recommended reading for everyone in medicine, from the junior medical student to the established practitioner.—*Marion E. Alberts*, M.D.

METHODS OF NEURO-OPHTHALMOLOGIC EXAMINATION, by *Alfred Kestenbaum*, M.D. (New York, Grune & Stratton, 1961. $16.75).

This is the second edition of a book that was originally published in 1946.

Every chapter has been expanded, and the book is now almost twice its original size. There have been some notable additions—specifically, the chapters on electroretinography and electromyography.

A number of improvements have been included in the examinations, notably in perimetry.

This book is a complete reference work and should be included in the library of any ophthalmologist who is interested in evaluating neurological findings.—*Henry H. Gurau*, M.D.

PRELIMINARY FINDINGS IN CANCER SOCIETY SURVEY

Nearly 40,000 human Iowa "guinea pigs" are currently participating in the most extensive cancer research project ever undertaken in the nation. This project, an epidemiological survey conducted by the American Cancer Society in twenty-five states, involves approximately 1,100,000 Americans from every walk of life, representing every race, creed and religion.

The six year project, now in its second year, is designed to evaluate family living habits and other environmental factors as they may relate to the incidence of cancer. Some environmental relationships are already known. For example, cancer of the skin is more prevalent in the southwestern United States, because of constant exposure to strong sunlight and winds. In the central part of the nation, gastrointestinal cancer is more common than in other areas. Other factors, such as exposure to certain chemicals, are known to have a relationship to the incidence of various types of cancer. Though these relationships are known, the degree to which they exist, and the possibilities of determining danger signals deserve further investigation.

The epidemiological survey in Iowa is under the direction of 2,500 American Cancer Society volunteers, who serve as "lay researchers" to distribute and collect the various confidential reports from participating persons. These confidential reports are not opened in the state, but are forwarded to the American Cancer Society's national headquarters in New York, where the statistical information is recorded on electronic tabulating machines.

Participants were originally enrolled by completing a comprehensive confidential questionnaire regarding their family living habits. Every 12 months, volunteer researchers check the status of all participants. Names of those who have died are forwarded to the Division of Vital Statistics of the Iowa State Department of Health. Death certificates are obtained, forwarded to national Society headquarters, and the physicians of those whose deaths were attributed to malignancies are asked to provide confidential reports regarding the characteristics of the disease as it existed.

As a result of the first year follow-up of Iowa participants in the fall of 1961, it was determined that there had been 229 deaths among participants, 56 of them directly attributed to cancer.

Nationally, preliminary investigation of survey records revealed some rather startling facts. Of all participants, 62.2 per cent of males and 54.9 per cent of females reported one or more complaints usually regarded as cancer danger signals. Furthermore, 30.5 per cent of the men and 25.1 per cent of the women reported two or more cancer danger signals.

Among subjects who reported one or more dan-

ger signals, only 11.9 per cent of the men and 17.6 per cent of the women reported that they had seen a doctor about the matter within the last year. However, the more severe the complaint, the greater was the percentage of subjects who reported having seen a doctor about it.

The proportion of subjects who reported seeing a doctor about complaints decreased with age.

Altogether 42.4 per cent of the men and 50.1 per cent of the women participating indicated that they "have a medical check-up regularly every year." The proportion doing so increased somewhat with the educational level.

Other valuable information, such as determination of the extent to which undiagnosed cancer exists among Americans, and the discovery of perhaps additional cancer danger signals which will make possible earlier detection, diagnosis and treatment, is also anticipated following completion of the six-year project.

The Iowa program, under the supervision of the American Cancer Society's Iowa Division, is approved by the Iowa Medical Society, and is being conducted in 23 Iowa counties. They are: Adair, Allamakee, Black Hawk, Cass, Cedar, Cerro Gordo, Cherokee, Clinton, Des Moines, Dubuque, Fayette, Hancock, Jasper, Linn, Madison, Mahaska, Marshall, Plymouth, Polk, East Pottawattamie, West Pottawattamie, Scott, Webster and Woodbury.

MEDICAL-LEGAL SEMINAR IN HONOLULU

The International Medical-Legal Society, a group founded to promote the interprofessional relations of doctors and lawyers, will hold its Second Postgraduate Seminar in Honolulu, February 19 through 23, 1962. A panel of physicians and attorneys has been selected to equip the attorneys with more adequate medical knowledge, and the physicians with a comprehensive review of the law of medical practice.

The costs of the round trip by plane and of accommodations at any of four Sheraton hotels in Honolulu vary from $832.50 to $962 per couple, starting from various West Coast embarkation points. Applications and a $100 deposit for reservations should be sent to Wm. P. Houser, M.D., the executive secretary of the society, 1206 South Eleventh Street, Tacoma 5.

The faculty is to include Joseph F. Sadusk, Jr., M.D., chairman of the AMA Committee on Malpractice; C. Joseph Stetler, LL.M., director of the AMA Legal & Socio-Economic Division; Ben Bernstein, LL.D., director of the Philadelphia Medical Legal Institute; Robert B. Allison, LL.B., chairman of the Medical Legal Committee, King County, Seattle; and several others. The program will allow ample time for sightseeing and other forms of entertainment, and several special events have been planned for the registrants' wives.

DOCTOR DIPLOMATS

Five physicians from Tulsa, Oklahoma, members of the First Presbyterian Church of Tulsa, are giving up their practices for six-week periods to serve voluntarily at the Miraj Medical Center in Miraj, India. Dr. C. S. Lewis, one of these five Tulsa physicians, recently reported to the AMA on the progress of the project labeled "Doctors in Asia."

The first of the group of volunteer physicians flew to Miraj in mid-August. He will return at the end of September, and the next doctor will make the trip. In all, the five physicians will donate a total of 30 weeks to the program. The project is endorsed by the Tulsa County Medical Society. Funds for medical equipment, transportation and other expenses were raised through church and public contributions.

Other groups of American physicians are also becoming interested in the possibility of initiating similar ventures in their own communities. For example, several doctors met with Dr. Lewis during his AMA visit to discuss the feasibility of adopting an overseas program which would provide medical care to another area of the world equally in need of such assistance.

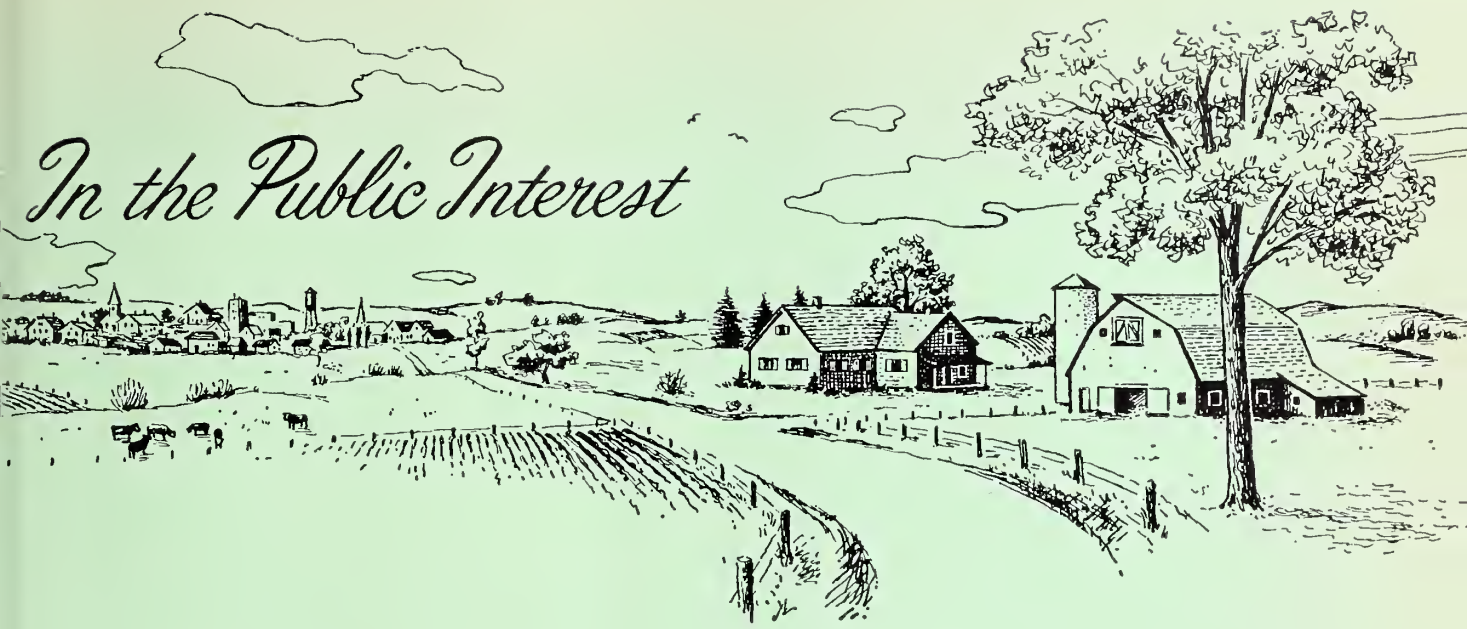
Still further evidence of American physicians' willingness to serve in foreign mission fields on a temporary basis is shown by the large number of doctors who have written to the AMA Department of International Health in the last few months to inquire about such service. This new Department administers a program approved last June by the AMA House of Delegates whereby members of the AMA may volunteer for service in the foreign mission fields on a temporary basis when emergencies arise. Cooperating with AMA in this program are missionary agencies representing every denomination sponsoring American medical missionaries.

Physicians interested in volunteering for such service are asked to write directly to the AMA Department of International Health, 535 N. Dearborn Street, Chicago 10, Illinois.

CHARTER FLIGHT TO EUROPE AND RETURN

The American Association of Automotive Medicine is making arrangements with Scandinavian Air Lines (SAS) for a charter flight to Europe and return sometime next spring. The round-trip fare will be approximately $220, and individual passengers will be free to spend four weeks in Europe altogether as they wish. The dates haven't yet been decided upon.

Inquiries should be addressed to Susan Snively, M.D., secretary, American Association of Automotive Medicine, Sacramento County Hospital, Sacramento.



Physicians and Government Will Work Together at

Protecting the Public From Medical Quackery

At a pioneering Conference on Medical Quackery, held in Washington, D. C., early in October, the enormity of the fake-cure and health-fad problem was outlined, in each of its several aspects, by a number of authoritative speakers, and some promising corrective measures were proposed. The sponsors of the gathering were the Food and Drug Administration, an agency of the federal government, and the American Medical Association, an organization of which 180,000 doctors of medicine are members.

Charlatanism in health care is by no means new, and both the physicians and the government have fought it for decades. Last month's conference, however, was evidence that the government and the medical profession have resolved henceforth to cooperate closely in that work, and to make a continuing educational campaign an important part of their joint endeavor.

There have been two principal reasons for the shortcomings of past attempts to protect the public from quacks. First, the help of the citizenry hasn't thus far been sought aggressively enough, and second, the laws in most states still require the critics of an unorthodox product or device to prove that it is either worthless or harmful, whereas the laws should require the proponents to prove that it is capable of doing what they say it will, and without producing any disastrous side effects. In the future, the AMA will work with federal officers, and the state and county medical societies will work with officials at their respective levels in remedying those deficiencies.

THREE TYPES OF FRAUDS

The speakers at the two-day Washington meeting talked about three types of frauds:

1. Expensive and useless treatments for medically incurable ailments, and diagnostic and treatment machines that do nothing more than dazzle the patient
2. Completely ineffective or merely palliative self-medications
3. Unnecessary dietary supplements.

Abuses of the first of those three types aren't so numerous as they were 25 years ago, in part at least because true physicians are meriting greater and greater degrees of confidence, and because there are fewer invariably fatal diseases than there used to be. Now that doctors of medicine can eradicate many early cancers by means of either surgery or x-ray, and can arrest early tuberculosis by means of drug therapy, the quack remedies for those ailments no longer have so lucrative a market. Much the same can be said about diabetes, the venereal diseases and several other ailments, and about the nostrums that were sold to people afflicted with them. But several of the major medical problems remain unsolved. Some people fail to seek competent care for tuberculosis or tumors until science can no longer help them. There are, as yet no cures for some of the malignancies, for any of several types of hereditary diseases, or for some much more common maladies such as arthritis, rheumatism and the common cold. The AMA reported at the Washington meeting that

Americans are wasting $250,000,000 annually on useless "cures" for arthritis and rheumatism, as one patient out of every two hunts in vain for a miracle, and that the cancer patients—most of whom have waited too long before seeking medical attention—are spending $50,000,000 each year for charlatans' treatments.

Lured by flamboyant advertisements and glowing promises, many people are putting themselves into the hands of "healers" of various sorts who are exceeding the scopes of practice that their states have granted them. These fakirs, in numerous instances, use electrical gadgets that they say will diagnose and/or treat human ills, though they consist of nothing more than blinking lights on impressive-looking panels. The federal or state governments, the audience in Washington was told, have stopped the manufacture of 30 or more brands of these machines, and have seized and destroyed the unsold stocks of them in the makers' warehouses. But the task of tracking down and destroying the models already in use has proved too expensive and time-consuming for the agencies' slender budgets and few investigators. Further, enforcing the licensing laws has been hampered by the fact that only the "healer" and his patient are present during a "treatment," and an investigator has difficulty in gathering convincing evidence of fraud, even when he is daring enough to let the suspect work on him.

No longer is there much of a market for "spring tonics" or the other high-priced fluids that once were advertised as cures "for whatever ails you," but the patent medicine business still thrives and frequently does a great deal of harm. Many products are offered as remedies for conditions that physicians are sure cannot be altered. These include progressive baldness in middle-aged men, minimal breast development in a considerable share of women, and the usual manifestations of the aging process in both sexes. Other patent medicines are do-it-yourself technics for achieving objectives that only a doctor can safely seek. These products are "guaranteed" to help the purchaser lose weight rapidly, remove moles or other skin blemishes, or escape pain in various parts of the body. But in losing weight quickly without medical supervision, he may lay himself open to an infectious disease; in removing skin blemishes, he risks disfiguring himself; and in masking pain he merely postpones a visit to his doctor—a visit that might reveal appendicitis, cancer or some other condition that could be fatal if long neglected.

People must be reminded again and again that the decision to buy and to take a non-prescription medicine constitutes self-diagnosis. Even when the product seems to help, it may be keeping them from seeking the definitive treatment that can, quite literally, mean the difference between life and death.

Nearly every one of the speakers at the Conference on Medical Quackery stressed the point that there is nothing wrong with the American diet, and attacked the varied theories of the food-faddists. Dr. Frederick Stare, of Harvard, said that adolescents—particularly teen-aged girls—frequently are undernourished, but that the fault can't be assigned to the food-processors or the farmers. The youngsters' mothers put adequate varieties and quantities of nutritious food on the table, but the young people simply refuse to eat it.

For the most part, nutritional quackery is merely economic foolishness. If one buys seaweed, "organically fertilized" vegetables and other esoteric foods, rather than the ones that crowd the shelves of every supermarket, he is wasting his money, but that's about all. If, however, he buys and eats these products in the expectation that they will relieve him of pain or some other symptom of disease, he is making a far more serious mistake. Furthermore, it is possible that an unbalanced diet of "health foods" can, paradoxically, *create* a nutritional deficiency.

Dr. Stare, one of the most prominent men in the field of nutrition research, apparently is unwilling to go along with the widely publicized theory that vitamin tablets are a waste of money. He said that though the majority of Americans are well fed, some individuals are not. "It would appear," he went on, "that the practicing physician is not likely to detect early deficiencies nor to be able to evaluate the vitamin intake accurately. . . . In this sense, vitamin supplementation is 'insurance' or can be considered a form of preventive medicine." He recommended that laymen look for the brands of vitamins marketed by major pharmaceutical manufacturers because those firms have quality-control procedures that help assure the buyer that he will get what he has paid for.

PHYSICIANS ARE ASKED TO TAKE ACTION

Doctors—individually and through their professional organizations—were urged by the speakers at the Conference to get their state legislatures to enable authorities more easily and promptly to seize, condemn and confiscate products or devices for which therapeutic benefits have been unjustifiedly claimed.

They were asked to assist their state and federal governments more actively in tracking down charlatans.

They were also asked to protest to the managers of radio and television stations about broadcasts in which "health food" salesmen charge that the soil has been depleted and that the standard food products available in grocery stores are nutritionally poor, and to protest about the broadcasts in which patent-medicine hucksters make extravagant claims for their products.

Finally, physicians can help if, when they have opportunities to address lay audiences, they will warn their listeners as specifically as possible about the types of quackery that they know to be rife in their particular communities.

INFORMED CONSENT

One of the most distinguished and respected judges in America, Chief Judge Benjamin Cardozo of the New York Court of Appeals, in 1914, summed up the whole subject of the law concerning consent to medical treatment in two brief sentences when he said:

> Every human being of adult years and sound mind has a right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient's consent commits an assault for which he is liable in damages. . . . This is true except in cases of emergency where the patient is unconscious and where it is necessary to operate before consent can be obtained.[1]

It is important to note that the legal theory under which a physician is sued to recover damages for treatment without consent is based not on malpractice but on assault. Even though the unauthorized procedure may have been skillfully performed and generally beneficial to the patient, the physician has committed, at least technically, an assault.

In a well-known Minnesota case illustrating this point, the patient had consulted a surgeon because of difficulty with her right ear. The surgeon advised surgery on the right ear for removal of a middle-ear polyp, and the patient consented to the procedure. While the patient was under anesthesia, the surgeon examined the left ear and found it in a more serious condition than the right ear. Thereupon he operated on the left ear. The Minnesota court in its well-reasoned decision held that:

> If [the surgeon's act] was unauthorized, then it was, within what we have said, unlawful. It was a violent assault, not a mere pleasantry; and, even though no negligence is shown, it was wrong and unlawful. The case is unlike a criminal prosecution for assault and battery, for there an unlawful intent must be shown. But that rule does not apply to a civil action, to maintain which it is sufficient to show that the assault complained of was wrongful and unlawful or the result of negligence.[2]

Not only must there be consent to medical treatment, but it must be an informed consent. The patient should have an understanding of what is to be done and of the hazards involved in the treatment. The physician should explain to the patient in understandable, nontechnical terms the procedure involved and its attendant risks.

In another Minnesota case, the patient had consented to a transurethral resection, but the surgeon had failed to advise him that it would be accompanied by a vasectomy. In holding that the question of assault should have been submitted to the jury, the court said:

> While we have no desire to hamper the medical profession in the outstanding progress it has made and continues to make in connection with the study and solution of health and disease problems, it is our opinion that a reasonable rule is that, where a physician or surgeon can ascertain in advance of an operation alternative situations and no immediate emergency exists, a patient should be informed of the alternative possibilities and given a chance to decide before the doctor proceeds with the operation. . . . Under such conditions the patient would at least have the opportunity of deciding whether he wanted to take the chance of possible infection if the operation was performed in one manner or to become sterile if performed in another.[3]

On the other hand, in an emergency where injuries require immediate surgery to preserve life, the surgeon may take such measures as he deems necessary.[4]

As stated by a Midwestern court:

> Only the operation specifically consented to may be performed unless the need for further surgery, essential to the preservation of the life or health of the patient, develops or becomes apparent during the performance of the surgery for which consent was given.[5]

But it should be always kept in mind that no separate, unrelated or entirely different operation should be performed.

In order for a consent to medical treatment or a surgical procedure to be valid, it must be the rational act of a qualified mind. The consent of the patient to the treatment or operation is sufficient if he has attained his majority and is, at the time of giving consent, competent to understand the nature and purpose of the treatment or operation proposed and the risks involved.

If the patient is a minor, the consent of the parent or guardian must be obtained. If the minor has parents, the consent should be obtained from them even though the minor is in the temporary charge of some other person. In the instance where the minors' parents are divorced or legally separated, consent for medical treatment or surgery should be obtained from the parent who has legal custody of the child.

The courts have held that even where there is an absolute necessity for some operation, such as prompt removal of a child's tonsils, but it is not an emergency in the sense that death would likely result immediately, an operation would not be justified without the parent's consent.[6]

However, as noted previously in Judge Cardozo's opinon, the courts recognize that when emergency treatment is required, an exception to the prohibition against unauthorized treatment or surgery is present. An Iowa court, for example, held that where a 17-year-old boy sustained a mangled elbow as the result of a fall from a freight train, the emergency justified the physician in proceeding with amputation without parental consent.[7]

The consent of the husband to an operation on his wife is not necessary; the consent of the wife is sufficient, and the converse is, of course, also true. Nevertheless, it is advisable to have the spouse join in the consent whenever practicable. It is particularly desirable to do so if the operation involves danger to life, may destroy or limit sex functions, or may result in the death of an unborn child.

It is usually mutually desirable for the phy-

sician to request that the patient allow him a reasonable amount of discretion in the performance of an operation.

Above all, it should be kept in mind that the operating physician, even in the case of a limited operation, must do whatever may be reasonably necessary for the safety of the patient.

REFERENCES

1. Schloendorf v. New York Hospital, 211 N. Y. 125.
2. Mohr v. Williams, 95 Minn. 261, 104 N.W. 12.
3. Bang v. Charles T. Miller Hospital, 251 Minn. 427, 88 N.W. 2d 186.
4. Sullivan v. Montgomery, 155 Misc. 448 (N. Y.); Luka v. Lowrie, 171 Mich. 122.
5. Paulsen v. Gundersen, 260 N.W. 448.
6. Banner v. Moran, 126 F. (2d.) 121; Moss v. Richworth, 222 S.W. 225 (Texas).
7. Jackovach v. Yocum, 212 Iowa 914, 237 N.W. 444.

—Editorial in the NEW YORK STATE J. MED., **61**:3412-3413, (Oct. 15) 1961.

25 COLLEGES AND UNIVERSITIES
PRODUCING THE LARGEST NUMBER OF ENTERING FIRST-YEAR MEDICAL STUDENTS
(1952-1960 IN TWO-YEAR INTERVALS)

1952		1954		1956		1958		1960	
School	No. of Ent. Med. Stud.	School	No. of Ent. Med. Stud.	School	No. of Ent. Med. Stud.	School	No. of Ent. Med. Stud.	School	No. of Ent. Med. Stud.
*Harvard	169	Harvard	174	Harvard	162	Harvard	182	Harvard	164
*Michigan	155	Michigan	136	Illinois	151	Michigan	164	Michigan	145
*Columbia	121	Illinois	105	Michigan	145	Illinois	121	Illinois	135
*Emory	98	Columbia	96	Columbia	145	Columbia	120	Columbia	131
*Indiana	96	Cornell	93	Wisconsin	122	Princeton	106	Princeton	120
*N. Y. U.	96	Yale	92	Cornell	107	Yale	101	Cornell	96
*Minnesota	96	Indiana	90	Yale	94	Penn.	98	Yale	96
*Illinois	95	Ohio State	90	Texas	91	Notre Dame	94	Wisconsin	93
*Yale	87	Cal. S. F.	90	Princeton	87	Cornell	94	Indiana	91
*Stanford	86	Minnesota	90	Cal. S. F.	87	Cal. S. F.	92	Dartmouth	91
*Princeton	85	Penn.	90	Minnesota	87	Wisconsin	87	Texas	89
*Penn.	83	Texas	84	Indiana	80	Dartmouth	84	Penn.	86
Pittsburgh	83	Wisconsin	79	Emory	78	Texas	83	Cal. S. F.	84
*Cal. S. F.	83	Emory	78	N. Y. U.	76	Cal. L. A.	82	Minnesota	84
*Wisconsin	82	Pittsburgh	78	Dartmouth	76	Stanford	81	Duke	83
*Texas	82	Princeton	76	Penn.	75	Emory	81	Stanford	81
Alabama	73	N. Y. U.	72	Stanford	74	Indiana	79	Ohio State	78
*Cornell	73	Stanford	70	Iowa	73	Minnesota	77	Cal. L. A.	73
Cal. L. A.	73	Alabama	70	Ohio State	72	Ohio State	76	Tulane	69
Kansas	71	Tulane	66	Cal. L. A.	71	Wayne	74	Wayne	69
Iowa	65	Dartmouth	65	Tennessee	68	N. Carolina	66	Emory	68
La. State	63	Duke	63	N. Carolina	64	Tulane	64	Iowa	63
Vanderbilt	62	Kansas	62	Notre Dame	62	Pittsburgh	62	Kansas	62
*Ohio State	61	N. Carolina	61	La. State	61	N. Y. U.	61	N. Y. U.	61
Duke	60	Holy Cross	59	Alabama	59	Duke	61	Notre Dame	56
Top 25 Schools Total	2,198		2,129		2,267		2,290		2,268
All Schools Total	7,381		7,424		7,835		7,925		8,075
Per Cent from Top 25 Schools	30%		29%		29%		29%		28%

* Among top 25 schools in all five years.

A relatively small number of colleges and universities continue to supply the majority of students admitted to U. S. medical schools each year. The accompanying table shows the 25 undergraduate institutions that supplied the largest numbers of entering first-year medical students in each of five recent years, and the numbers of students who came from each of them.

The table also shows the total numbers of entering first-year medical students each year, and the proportion of the entire entering freshman class supplied by the top 25 undergraduate schools.

Iowa Chapter of the American Academy of General Practice

REPORT ON THE ANNUAL MEETING

The Thirteenth Annual Meeting of the Iowa Chapter of the American Academy of General Practice was held during a noon luncheon at the Savery Hotel, in Des Moines, on Monday, September 26. There were 86 members present—more than twice the attendance in any previous year. The business was dispatched in an orderly fashion, since all reports had been mimeographed in advance and had been given out to all the members when they registered.

According to the treasurer's report, the Chapter's income had been sufficient to meet all obligations, and its financial condition is the best that it has been for some time.

The Scientific Assembly Committee reported on the various meetings that had been held during the preceding year, and announced the schedule of meetings for the year to come. The schedule of future scientific sessions is as follows:

Nov. 1, 1961—Burlington, with the Des Moines County Medical Society
Nov. 15, 1961—Sioux City, with the Woodbury County Medical Society
Feb. 13-16, 1962—Iowa City, Annual Refresher Course, with the S.U.I. College of Medicine
Mar. 15, 1962—Sioux City—Lederle Symposium
Apr. 9-12, 1962—Las Vegas—AAGP Annual Scientific Assembly
June 18-21, 1962—Okoboji—Spring Conference
Sept. 12-13, 1962—Des Moines—Fourteenth Annual Meeting of the Iowa Chapter

These meetings will provide a total of approximately 80 hrs. of Category I credit.

The Membership Committee reported a net increase of seven members. Our membership now totals 677. The four deaths that occurred during the fiscal year were those of:

Ruth F. Wolcott, M.D., Spirit Lake
Ralph Weaver, M.D., Cumberland
Robert C. McGeehon, M.D., Indianola
G. W. Rimel, M.D., Bedford

A membership drive is being started in an effort to enroll other eligible physicians. Dr. Donald Kast, of Des Moines, is chairman of the National Membership Committee of the AAGP, and of course is interested in seeing the Iowa Chapter improve its membership standing.

The General Practice Committee reported that talks had been held with representatives of the S.U.I. College of Medicine during the year, and that those meetings had helped to widen and deepen the relationship between the Academy and the medical school.

The Public Relations Committee announced that the Iowa Chapter is again going to sponsor the "Ask the Doctor" program over the KRNT radio station in Des Moines, beginning on October 4. The series is scheduled for every Wednesday night at 8:30 for 13 weeks. Members wishing to appear on the program were asked to call the Iowa Chapter office.

The following new officers were elected:

Eugene Smith, M.D., Waterloo, president-elect
Wm. A. Castles, M.D., Dallas Center, vice-president
Arnold T. Nielsen, M.D., Ankeny, secretary-treasurer
George A. Paschal, M.D., Webster City, member of board of directors
John R. Jaquis, M.D., Reinbeck, member of board of directors
C. H. Stark, M.D., Cedar Rapids, delegate to the AAGP
R. L. Bartley, M.D., Audubon, alternate delegate to the AAGP

On Monday evening a reception, buffet dinner and dance were held. Dean Norman B. Nelson, of the S.U.I. College of Medicine, spoke following the dinner, and in a forceful way illustrated the need for close cooperation among medical organizations. If medical schools and organizations of doctors such as the AAGP "do not work together, they will go down together," he declared.

There were 36 technical exhibitors whose financial assistance made the meeting possible. The scientific lectures were highly informative and well received, setting a high standard in the field of postgraduate education.

POSTGRADUATE CONFERENCE

Sheraton-Martin Hotel, Sioux City

Wednesday, November 15

1:00 p.m. Registration
2:00 p.m. "Spontaneous Pneumothorax and Cardiac Arrest"—Harry H. McCarthy, M.D., Omaha
2:40 p.m. "Endometriosis"—John L. McKelvey, M.D., head of obstetrics and gynecology, University of Minnesota Medical School
3:20 p.m. "Cardiac Arrhythmias"—Herman J. Smith, M.D., Des Moines
4:20 p.m. "Surgical Lesions of the Lung—Diagnosis and Treatment"—Dr. McCarthy
5:00 p.m. "Bleeding in the Last Trimester"—Dr. McKelvey
5:40 p.m. "The Medico-Legal Aspects of Heart Disease"—Dr. Smith
6:30 p.m. SOCIAL HOUR AND DINNER
8:00 p.m. PANEL DISCUSSION—All speakers

This program has been made possible by a grant to the Iowa Chapter of the AAGP by Eli Lilly and Company. Five hours of Category I credit will be allowed Academy members for attendance at this meeting.

POSTGRADUATE SHORT COURSES AT IOWA CITY

On November 8 and 9, at University Hospitals in Iowa City, there will be an Institute on Intrapartum and Neonatal Complications sponsored by the College of Medicine's Departments of Obstetrics and Gynecology and Pediatrics, the S.U.I. College of Nursing, and the Division of Maternal and Child Health of the State Department of Health.

A postgraduate course "Current Concepts in the Diagnosis and Treatment of Cardiovascular Diseases" will be held on November 17-18 at University Hospitals. This course is sponsored by the Iowa Heart Association and the S.U.I. Department of Internal Medicine.

Registration forms may be obtained by writing to John A. Gius, M.D., Director, Postgraduate Medical Studies, College of Medicine, Iowa City. The program schedules for the two courses follow.

INSTITUTE ON INTRAPARTUM AND NEONATAL COMPLICATIONS

Wednesday, November 8, 1961

8:30 Registration
Moderator: W. C. Keettel, M.D., S.U.I.
9:00 Welcome—Dean Norman B. Nelson, S.U.I.
9:10 Cause and Detection of Fetal Distress
W. C. Keettel, M.D.
9:40 The Nurse's Role in Recognition of Fetal Distress
Agnes McConnell, R.N., Regional Nursing Consultant, Children's Bureau, Kansas City, Mo.
10:00 Resuscitation of the Newborn
C. P. Goplerud, M.D., S.U.I.
10:45 Respiratory Distress and Its Recognition
Herbert Miller, M.D., Head of the Department of Pediatrics, University of Kansas
11:15 Anoxia and Its Effect Upon the Newborn
Donal Dunphy, M.D., S.U.I.
11:45 General Discussion With Questions
12:30 Luncheon—Doctors' Dining Room

PROGRAM FOR PHYSICIANS

2:00 to 3:30 p.m. Small Group Discussions
Prophylaxis and Management of Habitual Abortion—J. P. Jacobs, M.D., S.U.I.
Neonatal Jaundice—R. E. Carter, M.D., S.U.I.
Induction of Labor—W. F. Howard, M.D., S.U.I.
Convulsions of the Newborn—John MacQueen, M.D., S.U.I.

PROGRAM FOR NURSES

Moderator: Gladys Benz, R.N., S.U.I.

2:00 Use of Ancillary Help in Labor and Delivery Room—Doris Wilkinson, R.N., S.U.I.
2:30 Simplified Postpartum Care—Leone Johnson, R.N., Nurse Consultant, Division of Maternal and Child Health, Iowa State Department of Health
3:00 Prenatal Classes—Anna Overland, R.N., and Muriel Raines, R.N., S.U.I.
4:00 Combined Conference, Prevention of Nursery Infections
Madelene Donnelly, M.D., Director, Division of Maternal and Child Health, State Department of Health
Donal Dunphy, M.D.
Herbert Miller, M.D.
Gladys Benz, R.N.
6:30 Dinner, Iowa Memorial Union—Dr. Donnelly, presiding
Guest Speaker—Dr. Frederick Falls, Professor Emeritus, University of Illinois College of Medicine
"Cooperative Management of Obstetrical Problems"

Thursday, November 9, 1961

Moderator: Donal Dunphy, M.D.

9:00 Spontaneous Premature Rupture of Membranes
Obstetrical Management—C. A. White, M.D., S.U.I.
Pediatric Management—Herbert Miller, M.D.
9:30 Symposia on Prematurity
Cause and Prevention—F. H. Falls, M.D.
Management of Premature Labor—C. W. Seibert, M.D., Waterloo Pediatrician
Nursery Supervision—Sister M. Zita, R.N., Director, Department of Obstetrics, Mercy Hospital, Des Moines
Pediatric Problems—Charlotte Fisk, M.D., Des Moines Pediatrician
Followup—Madelene Donnelly, M.D.
11:00 to 12:00 Perinatal Mortality Conference, Methodist Hospital, Des Moines—G. G. Caudill, M.D., Des Moines Pediatrician
12:30 Luncheon—Doctors' Dining Room

CURRENT CONCEPTS IN THE DIAGNOSIS AND TREATMENT OF CARDIOVASCULAR DISEASES

Friday, November 17, 1961

8:15 Registration
8:50 Introductory Remarks

9:00 Treatment of Severe Myocardial Infarction
Lewis E. January, M.D., S.U.I.
9:35 X-Ray Diagnosis of Cardiac Disease With Conventional Films
Eugene F. Van Epps, M.D., S.U.I.
10:30 Right Heart Failure and Pulmonary Disease
George N. Bedell, M.D., S.U.I.
10:05 Digitalis Intoxication
Ernest O. Theilen, M.D., S.U.I.
11:40 *Panel Discussion:* Present Day Treatment of Hypertensive Patients: Who, When, How?
Walter M. Kirkendall, M.D., S.U.I.; Victor E. Pollak, M.D., Research Assistant Professor of Medicine, University of Illinois College of Medicine; Mark L. Armstrong, M.D., S.U.I.
12:30 Lunch
2:00 Hypertension in Pregnancy
Victor E. Pollak, M.D.
2:35 The Pharmacologic Basis for the Action of Diuretics
Walter M. Kirkendall, M.D.
3:30 Renal Changes in Collagen Vascular Disease
Victor E. Pollak, M.D.
4:05 *Panel Discussion:* Fluid and Electrolyte Management in the Patient With Impaired Renal Function
Mark L. Armstrong, M.D.; Edward E. Mason, M.D., S.U.I.; George M. Owen, M.D., S.U.I.

Saturday, November 18, 1961

9:00 Congestive Heart Failure in Infancy
Jacqueline A. Noonan, M.D., S.U.I.
9:30 Thrombosis and Thrombolysis
Anthony P. Fletcher, M.D., Assistant Professor of Medicine, University of Illinois College of Medicine
10:30 The Effect of Drugs on the Microcirculation
Frank M. Abboud, M.D., S.U.I.
11:05 *Panel Discussion:* Treatment of Occlusive Arterial Disease: Vasodilators, Anticoagulants, Thrombolytic Agents, Sympathectomy and Vascular Surgery
John W. Eckstein, M.D., S.U.I.; Montague S. Lawrence, M.D., S.U.I.; Anthony P. Fletcher, M.D.
11:50 The Effect of Anesthetic Agents and Other Drugs on the Heart
Charles B. Pittinger, M.D., S.U.I.

TREATMENT OF MENTAL RETARDATION BY THE GP

A short course entitled "Practical Considerations in the Treatment of Mental Retardation by the General Practitioner" will be presented in the Auditorium of the Nebraska Psychiatric Institute, in Omaha, on Thursday, November 16. The registration fee will be $15.

A film on phenylketonuria will be shown, and there will be panel discussions of (1) the scope of the mental retardation problem, (2) diagnostic considerations, (3) the mental retardation research center, and (4) practical considerations of mental retardation in general practice. For further information, address the Division of Postgraduate Affairs, University of Nebraska College of Medicine, Forty-second and Dewey Avenue, Omaha 5.

AMA DUES INCREASE

The House of Delegates of the AMA approved an increase in annual dues at its meeting in New York City last June. The action called for an increase of $10 on January 1, 1962 and an additional increase of $10 on January 1, 1963. The House disapproved of ear-marking any portion of the increase in dues for any specific purpose. The dues for 1962 will total $35, and for 1963, $45.

Among the new and expanded AMA programs for which the additional income will be used are:

- A far-reaching new drug information program, an enlarged program for a drug-standards laboratory, and a cooperative program for the selection of nonproprietary names for drugs.
- A complete study of internships and residencies in the U. S. to determine (a) the specific purposes to be achieved by graduate medical education; (b) the ideal design of educational programs to achieve those purposes; (c) plans for implementing that design as quickly as possible.
- A study of immediate problems related to internship programs, and the framing of recommendations to balance the number of internships with the number of internes available to fill them, and to help hospitals find ways of getting internes' work done by other personnel.
- A recruitment program to attract more talented students into the study of medicine, a student honors program, and financial assistance to medical students.
- A vigorous effort to combat mental illness.
- An educational program for the public, stressing healthful living habits, physical fitness for young people, traffic safety, reduction of air and water pollution, preventive medicine, and the elimination of misleading advertising of health care products.
- An international program in cooperation with world medical organizations to assist medical missionaries and bring better health to people everywhere.

REOPENING OF IMS INSURANCE GROUP

The statewide Blue Cross-Blue Shield group program for physicians that was approved by the IMS Committee on Group Insurance and by the IMS House of Delegates will complete two years of operation in January, 1962.

This plan for IMS members is being reopened for new enrollments and transfers. Applications will be accepted prior to December 1, 1961. For doctors having Blue Cross and Blue Chip coverage, no increase in the overall rate will be necessary for the coming year. This means a third year of coverage without an increase in rate.

Information on the provisions of this plan has recently been mailed to all physicians who have not as yet availed themselves of it.

STATE DEPARTMENT OF HEALTH

Edward G. Zimmer

COMMISSIONER

MORBIDITY REPORT FOR MONTH OF SEPTEMBER 1961

Disease	1961 Sept.	1961 Aug.	1960 Sept.	Most Cases Reported From These Counties
Diphtheria	1	0	0	Polk
Scarlet fever	95	44	42	Johnson, Polk
Typhoid fever	1	1	1	Woodbury
Smallpox	0	0	0	
Measles	23	63	18	Buena Vista, Scott
Whooping cough	4	1	6	Scott
Brucellosis	11	10	12	Dubuque, Scott
Chickenpox	28	20	9	Buena Vista, Dubuque, Scott
Meningococcic meningitis	0	0	1	
Mumps	35	65	108	Buena Vista, Dubuque, O'Brien, Polk, Scott
Poliomyelitis	4	6	3	Audubon, Clay, Kossuth, Scott
Infectious hepatitis	139	95	29	Polk, Pottawattamie, Scott, Warren
Rabies in animals	32	32	7	Jackson, Johnson, Keokuk, Washington
Malaria	0	0	0	
Psittacosis	0	0	0	
Q fever	0	0	0	
Tuberculosis	24	25	30	For the state
Syphilis	109	83	71	For the state
Gonorrhea	156	109	89	For the state
Histoplasmosis	4	1	0	Dallas, Polk, Wapello
Food intoxication	0	0	0	
Meningitis (type unspecified)	34	12	3	Clay
Diphtheria carrier	0	0	0	
Aseptic meningitis	13	0	9	Polk, Warren
Salmonellosis	1	5	2	Dubuque
Tetanus	0	1	0	
Chancroid	0	0	0	
Encephalitis (type unspecified)	1	0	3	Carroll
H. influenzal meningitis	0	0	0	
Amebiasis	3	3	1	Boone
Shigellosis	3	2	10	Lee, Webster, Woodbury
Influenza	10	0	7	Clay

GONORRHEA AND SYPHILIS IN IOWA

A recent analysis of gonorrhea cases reported in Iowa (see chart on the next page) shows that 56.9 per cent of cases for which the patients' ages were specified fell in the 15-24 year age range. These figures indicate that gonorrhea poses a youth problem.

A comparison of the total cases reported in 1957 (598) and in 1960 (1,401) makes it appear that the incidence is increasing. Intensified case-finding and better reporting undoubtedly have helped to make the picture somewhat unduly alarming, but the increasing number of requests for assistance from the State Department of Health in resolving local problems or combatting epidemics indicates that gonorrhea is still a public health problem.

The second chart, showing syphilis morbidity in Iowa by age groups, was made primarily for comparison between the ages of patients reported as having gonorrhea and of those reported as having syphilis. Of the gonorrhea cases reported for the 10-year period, 19.86 per cent were in people between 15 and 19 years of age, 30.09 per cent were in the 20-24 year group, and 17.62 per cent in the 25-29 year group. That distribution contrasts sharply with the distribution of syphilis cases, for more syphilis cases have been reported in patients between 50 and 59 years of age.

We know that most people don't *get* syphilis that late in life, and though we are convinced that there is much more teen-age gonorrhea than syphilis, we know too that gonorrhea is reported rather promptly after the patient has contracted the infection. Contrastingly, over 95 per cent of reported cases of syphilis are non-infectious, and we can assume that the patients were infected many years earlier. Some of the cases had been diagnosed and treated, but not reported until quite recently. In many instances, however, the infections were contracted long ago, but the disease remained unrecognized until the year of reporting.

Nationally, there has been an increase in the incidence of early syphilis since 1955, but that trend hasn't yet been shown in Iowa.

Help your central office to maintain an accurate mailing list. Send your change of address promptly to the JOURNAL, 529-36th Street, Des Moines 12, Iowa.

GONORRHEA MORBIDITY IN IOWA BY AGE GROUPS, SEX, YEAR REPORTED AND PERCENTAGE EACH AGE GROUP REPRESENTS OF TOTAL MORBIDITY FOR THE ELEVEN YEAR PERIOD, 1950-1960

Age Group	Sex	1950	1951	1952	1953	1954	1955	1956	1957	1958	1959	1960	Total	Percent
Infants	M		1			1							2	.10
1 Year	F	1	1						1	2		2	7	
1-4 Years	M											1	1	.12
	F		1	1		1			4	1	1	1	10	
5-9 Years	M												0	.20
	F	2	3	1		2			1	6	3		18	
10-14 Years	M		7		1		1	1	1		1	3	15	1.76
	F	8	4	10	32	15	3	12	13	17	11	15	140	
15-19 Years	M	50	47	38	59	52	45	45	43	63	83	119	644	19.86
	F	53	77	70	130	101	74	114	70	96	138	185	1108	
20-24 Years	M	151	113	86	129	118	102	129	110	175	224	333	1670	30.07
	F	71	68	63	77	88	69	81	66	126	109	164	982	
25-29 Years	M	89	96	103	96	84	100	87	55	109	94	191	1104	17.62
	F	32	47	26	25	33	32	44	30	52	52	77	450	
30-39 Years	M	72	50	47	49	69	48	63	61	92	108	138	797	13.16
	F	37	28	24	27	35	19	26	24	31	57	56	364	
40-49 Years	M	25	15	18	13	23	20	18	15	17	26	33	223	3.54
	F	13	5	3	2	8	10	7	2	14	17	8	89	
50-59 Years	M	6	4	1	11	8	6	4	2	3	7	9	61	.92
	F	2	4	2		1	4	1		2	3	1	20	
60-69 Years	M	4	3	1	1	1	1		1	1	4	3	20	.27
	F			1			1	1			1		4	
70-79 Years	M		2								1		3	.05
	F										1		1	
80 Plus	M	1											1	.01
	F												0	
Age Not Stated	M	105	49	57	81	97	69	73	72	88	51	48	790	12.31
	F	29	31	18	36	44	31	16	27	28	22	14	296	
Sub-Totals	M	503	387	351	440	453	392	420	360	548	599	878	5331	60.44
	F	248	269	219	329	328	243	302	238	375	415	523	3489	39.55
Grand Total		751	656	570	769	781	635	722	598	923	1014	1401	8820	

SYPHILIS MORBIDITY IN IOWA BY AGE GROUPS, SEX, YEAR REPORTED AND PERCENTAGE EACH AGE GROUP REPRESENTS OF TOTAL MORBIDITY FOR THE ELEVEN YEAR PERIOD, 1950-1960

Age Group	Sex	1950	1951	1952	1953	1954	1955	1956	1957	1958	1959	1960	Total	Percent
Infants	M	10	5	2	2	3	3			2		1	28	.41
1 Year	F	5	6		5	3	6	1	2		3	2	33	
1-4 Years	M	1	1		1	1				1			5	.07
	F	1	3		1								5	
5-9 Years	M	1	2		3	1	1			1			9	.08
	F	1		1		1							3	
10-14 Years	M	9	6	3	2	1	3		1			1	26	.40
	F	11	6	6	2	5	2			1		1	34	
15-19 Years	M	25	28	17	23	15	11	13	9	6	8	3	158	3.03
	F	56	55	19	47	27	19	27	15	12	9	8	294	
20-24 Years	M	79	59	23	37	31	19	22	24	15	23	18	350	5.52
	F	93	69	52	65	50	35	23	27	24	18	19	475	
25-29 Years	M	78	75	36	35	28	14	17	19	12	9	18	341	5.74
	F	90	75	60	71	53	38	27	32	23	23	24	516	
30-39 Years	M	147	125	78	123	82	48	36	51	55	41	29	815	13.28
	F	193	160	95	180	121	87	73	78	62	55	65	1169	
40-49 Years	M	213	169	129	198	161	90	81	97	104	90	63	1395	19.80
	F	193	144	112	222	192	114	106	136	131	109	103	1562	
50-59 Years	M	188	194	115	201	190	131	135	147	147	131	125	1704	20.31
	F	127	108	88	170	144	90	111	113	143	111	125	1330	
60-69 Years	M	139	161	83	133	111	106	92	153	139	134	140	1391	14.99
	F	61	55	56	94	76	79	55	73	108	101	91	849	
70-79 Years	M	29	28	30	23	43	30	23	57	79	88	76	506	5.62
	F	16	9	14	15	21	32	12	42	55	56	62	334	
80 Plus	M	2	1	6	3	12	9	11	12	18	14	17	105	1.16
	F		1	4	2	3	2	6	14	14	11	11	68	
Age Not Stated	M	62	59	48	63	67	61	48	79	70	56	64	677	9.58
	F	55	66	51	114	88	69	66	81	76	48	40	754	
Sub-Totals	M	983	913	570	847	746	526	478	649	649	594	555	7510	50.28
	F	902	757	558	988	784	573	507	613	649	544	551	7426	49.72
Grand Total		1885	1670	1128	1835	1530	1099	985	1262	1298	1138	1106	14, 936	

Iowa Association of Medical Assistants

MIND YOUR BUSINESS

The following is the sort of welcome that representatives of Iowa Medical Service (Blue Shield) and of the Iowa Medical Society should receive when they visit the offices where we work: "Good morning, Mr. Jones. I'm glad you called today. I have a few questions that I have been saving for your next visit, and Dr. Smith has a problem to discuss with you. I think we can clear up my questions by the time Dr. Smith comes in, and then he will have time to talk with you while I am running some laboratory tests on his first patient." Yet, how often do they hear it? All too rarely!

All of us tend to criticize the organizations that we belong to and work with, and to find fault with the actions they take on particular issues. We grumble to ourselves and to others, but we fail to voice our objections and to make suggestions where they will do the most good. Physicians are similarly inclined, and we sometimes are partially responsible. Whenever we can, we should enable the field representatives of these organizations to visit with our doctors, so that those physicians can have a voice in deciding how the affairs of organized medicine are conducted.

The Iowa Medical Society and Iowa Medical Service (Blue Shield) belong to and should be operated by the physicians of the state. The field secretaries provide a means by which they can transmit their attitudes and their suggestions, and by which they and we can keep informed about changes of policy, legislative actions that are pending, and IMS, Blue Shield and AMA projects.

Sometimes we take our job of maintaining an appointment schedule, or conserving our employers' time, so seriously that we neglect to consider how important the field secretary's call can be to us in our work and to the doctor in his work.

Scheduled appointments, of course, must take precedence over callers. It is obvious that a patient would resent having to wait beyond the time of her appointment while the doctor or the medical assistant passed the time of day (as she sees it) with a caller who came in after she did and who obviously did not have an appointment. In some instances the field secretary is traveling on a fairly flexible schedule and can wait a few minutes or come back later for a visit with you and the doctor. At other times, however, he can't easily wait or come back, and we should make every effort to fit him in. Perhaps he might be able to let you know in advance and can ask you to save some time for him.

We sincerely suggest that you discuss this matter with your employer, just as you would any other office policy, and that you discuss it with other medical assistants at one of your chapter meetings. The talks given by IMS and Blue Shield representatives at meetings of Medical Assistant Societies have been very helpful. If your chapter hasn't had one, we suggest that you request a speaker for a future meeting.

ANNUAL IN-SERVICE WORKSHOP

When plans were made in the early spring of this year for the third annual In-Service Workshop at SUI, we chose a week in early September. But faculty changes and vacations made it necessary for us to make some changes in the plans. The only other time available on the 1961 SUI schedule was September 25-27, so the course was shortened to three days. Apparently that arrangement was more acceptable than the earlier one, for preregistration boomed. The registration of 50 had been filled six weeks before the course was to start, and subsequent applicants were placed on a waiting list in case there were cancellations.

On Sunday afternoon, September 24, registrants checked in at the SUI Continuation Center and then were taken by SUI bus to Bill Zuber's restaurant in Homestead for a delicious Amana-style dinner. There, Dr. G. H. Scanlon, of Iowa City, talked briefly on "What the Doctor Expects of His Medical Assistant."

Three busy days of classes began on Monday. Professor W. R. Hudson, of the College of Engineering, discussed "Simplified Patterns of Work Flow," and Dr. Ralph Ojeman of the Iowa Child Welfare Research Station, talked on "Child Psychology." Dr. Coder conducted classes on "The Importance of Proper English Usage" and "How to Spell." Professor Cleo Casady, of the College of Business Administration, took up "Business Letters," and Miss Edith Ennis, of the same department, discussed "Reception Techniques and Appointment Making." Professor Jack Flagler, of the Bureau of Labor and Management, discussed "Human Behavior and Its Causes." Miss Dorothy Vogel, of the Northwestern Bell Telephone Company, completed the classes by demonstrating "Proper Use of the Telephone."

Leisure hour entertainment was provided by the Iowa City chapter. On Monday evening, the registrants were taken on tours of local offices to inspect accounting systems, filing and records equipment and room arrangements. On Tuesday evening, the chapter entertained at its monthly meeting, and its medical advisors and their wives were guests, too. Following dinner at the Mayflower Inn, Dr. Harold Mulford, a psychologist on the staff of the SUI Department of Psychiatry, spoke on "Alcoholism."

Woman's Auxiliary News

OUR PRESIDENT SAYS—

November is the month for our Thanksgiving—an institution born on American soil in 1621, when the Puritans of Massachusetts set apart a day to be thankful for their many blessings and freedoms. This year let's not only give thanks for our blessings but also start redoubling our efforts to preserve the liberties that have been our heritage.

The Eighteenth Annual Conference for state Auxiliary presidents, presidents-elect, national officers and committee chairmen convened at the Drake Hotel, in Chicago, October 1-4. I am happy to report that Mrs. A. C. Richmond, the first vice-president, Mrs. R. F. Nielsen, a member of the Committee to Study International Philanthropic Interests, Mrs. E. A. Larsen, the north-central regional chairman of Rural Health, and Mrs. Hazel Lammey, our administrative secretary, attended that fine conference with me. We had an opportunity to meet the officers from other states, all endeavoring to develop Auxiliary projects at the county level. We have returned to our homes eager to promote better Auxiliary understanding, proud to be Auxiliary members, for we have a unique and important role as wives of physicians to "put our shoulders to the wheel," as Mrs. Harlan English expressed it.

As Osler said, "We are commanded by our God to be servants of life as long as we live." In that service, let us speak out honestly for medicine. That we can do so is a privilege we should be thankful for.

I regret to announce the resignation of our president-elect, Mrs. E. B. Dawson, but life brings uncertainties, and they must be accepted and respected. We have appreciated your fine endeavors in Auxiliary work, Paulyne, and we shall look forward to your returning to participation. Take heart, each one of you! Mrs. Richmond, our first vice-president, has agreed to accept the post that Mrs. Dawson vacated. She was elected and installed by the Board of Directors on October 24, 1961. This move created a vacancy in the office of first vice-president, and the Board elected Mrs. C. A. Trueblood, of Indianola, as first vice-president and chairman of Membership. We appreciate the loyalty and interest exhibited by these fine women in accepting greater responsibilities for our state Auxiliary.

Thanks to each of the councilors and members who helped arrange the splendid district meetings that were held in September and October.

—Mrs. Benj. F. Kilgore
President

HAVE YOU HEARD?

Hearings on H.R. 4222 (King-Anderson Bill), by the House Ways and Means Committee began on July 24 and continued through August 4. Over 400 persons requested an opportunity to appear, of whom 119 witness were granted the opportunity. The Iowa Medical Society requested an opportunity to testify at the hearings but this request was not granted. However, H. E. Wichern, M.D., chairman of the IMS Legislative Committee, personally filed a statement that will be entered in the record of the hearings. It was also printed in the October issue of the JOURNAL OF THE IOWA MEDICAL SOCIETY.

Seventy witnesses appeared in opposition to the bill. These included state and county medical societies, SAMA, AMA, and individual physicians as well as representatives from the Farm Bureau, Chamber of Commerce, Association of Manufacturers and allied medical groups.

Those who testified in favor of the bill included Abraham Ribicoff, Secretary of HEW, representatives of settlement and welfare agencies, American Nurses Association, Golden Age Clubs, American Public Welfare Association, Social Democratic Federation, Socialist Party, YWCA, AFL-CIO and Walter Reuther of the United Auto Workers whose testimony took up the better part of an entire day.

Seventeen (of the total 25 membership) members were present at the Ways and Means Committee hearings on the days when testimony was presented by Drs. Larson and Annis and also during the testimony of Walter Reuther.

Pertinent questions were asked throughout the hearings by Representatives Curtis of Missouri and Alger of Texas. Together with Congressmen Knox of Michigan, Utt of California and Betts of Ohio they criticized many of the witnesses who appeared in support of the bill for their failure to present the facts and for seeming to concentrate their efforts on attacking organized medicine.

Cecil King of California, sponsor of the bill, was the prime interrogator and was in constant attendance.

On the oral presentations, 45 witnesses appeared for the bill and 70 against. In the filed written statements, approximately 78 were in favor of the King bill and 165 against.

A strong push will be made for enactment of the King-Anderson Bill when Congress resumes in January. President Kennedy has stated that he will assign top priority to the Administration's social security medical plan for the aged. Administration strategists are counting on the fact that next year being an election year, lawmakers will

be more likely to support the bill if they are facing an immediate election contest. It is this strategy of using legislation for personal, political expediency that has resulted in the vastly expanded benefits and coverage of the social security program.

Now is the time to work if socialized medicine is to be defeated. Contact your county medical organization and see what you can do to help. Participate in "Operation Coffee Cup." Be informed—inform others.

—MRS. HOWARD G. ELLIS
Legislative Chairman

BOOKS FOR MEDICAL MISSIONARIES

At the 1961 Convention of the AMA Auxiliary, Mr. J. Raymond Knighton, of Oak Park, Illinois, executive director of the Christian Medical Society, asked our help in securing reference material for medical missionaries in the far corners of the world. Over 500 doctors are working in primitive situations and lack basic medical textbooks. They need books less than five years old, for the older ones are out of date. Medical libraries that are kept current are a possible source.

Following are a few of the books that have been requested and the dates of the most recent editions of some of them:

Cecil, TEXTBOOK OF MEDICINE, Tenth Edition, 1959
Conn, CURRENT THERAPY, 1961
Davis, CHRISTOPHER'S SURGERY
Greenhill, OBSTETRICS, Twelfth Edition, 1960
Campbell, UROLOGY, 3 vols., 1954
Ballenger, DISEASES OF THE E., N. & T., Tenth Edition, 1957
Willson, Beecham, Forman and Carrington, OB. & GYN.
Campbell, OPERATIVE ORTHOPEDICS, Second Edition, 2 vols.
Manson, TROPICAL DISEASES, Fifteenth Edition, 1960
Mitchell & Nelson, TEXTBOOK OF PEDIATRICS, Sixth Edition
Duncan, DISEASES OF METABOLISM, Fourth Edition, 1959
Collins, PRINCIPLES AND PRACTICES OF ANESTHESIOLOGY
Wintrobe, HEMATOLOGY, Fifth Edition, 1961
Beckman, PHARMACOLOGY, 1961
Gross, SURGERY OF INFANCY AND CHILDHOOD
Higgins & Orr, OPERATIONS OF GENERAL SURGERY, Third Edition, 1958
Meschan, ROENTGEN SIGNS IN CLINICAL DIAGNOSIS, 1956
Goodman and Gillman, PHARMACOLOGICAL BASIS OF THERAPEUTICS, Second Edition, 1955
Pillsbury *et al.*, MILITARY MEDICAL MANUAL (DERMATOLOGY) 1942
Gifford, TEXTBOOK OF OPHTHALMOLOGY, Sixth Edition, 1957
Sollman, MANUAL OF PHARMACOLOGY
Thorek, SURGICAL ERRORS AND SAFEGUARDS, Fifth Edition, 1960
Greenhill, OFFICE GYNECOLOGY, Seventh Edition, 1959
X-RAY (6 vols.), Yearbook Publishers

Get further information from the president of your county Auxiliary, or from Mrs. Wm. B. Chase, 690 Sixty-third Street, Des Moines 12. Members-at-large may send books to Mrs. Chase for mailing to the Christian Medical Society.

DISTRICT MEETINGS

District V

The members of the Fifth District of the Woman's Auxiliary to the Iowa Medical Society met at the Hotel Holst, in Boone, for a luncheon on Monday, September 18, with members from seven counties in attendance. Dr. Ben T. Whitaker, a past-president of the Iowa Medical Society, addressed the group briefly, stressing the importance of the Auxiliary in medicine's program. Dr. Ralph Wicks, of Boone, a member of the IMS Legislative Committee and a legislative contact man, discussed proposed laws that would affect medical care.

Mrs. H. W. Smith, councilor of the Fifth District, presided and introduced the state officers and committee chairmen. They told of the goals of the organization and of the work that is being accomplished. The officers present were: Mrs. B. F. Kilgore, state president, Des Moines; Mrs. E. B. Dawson, president-elect, Fort Dodge; Mrs. G. A. Paschal, parliamentarian, Webster City; Mrs. R. H. Moe, chairman of the Safety Committee, Griswold; Mrs. Einer Juel, co-chairman of Safety, Atlantic; Mrs. E. A. Vorisek, finance chairman, Des Moines; Mrs. N. A. Schacht, area chairman for the annual meeting, Fort Dodge; Mrs. O. C. Buxton, co-chairman of Rural Health, Webster City; and Mrs. Hazel T. Lammey, administrative secretary, Des Moines. The county presidents also reported on the projects that are being carried on in their respective Auxiliaries.

The projects that were discussed included: community service, handicapped craft sales, civil defense, safety, mental health, vision screening, leprosy relief, sending of medical magazines, books and pamphlets to medical missionaries, essay contest, and the sponsoring of future nurses and health careers clubs, as well as nurse recruitment. Another project is the support of the American Medical Education Foundation and of the Health Educational Loan Fund (formerly the Nurses Loan Fund).

It was noted that in the 10 years during which the Health Educational Loan Fund has been in existence, 48 students have been helped financially to attain their education in nursing or other health fields.

The Boone County Auxiliary was in charge of arrangements for the luncheon, the committee consisting of Mrs. L. R. Greco, president, Mrs. W. H. Longworth, Mrs. A. W. Puntenney, Mrs. R. L. Wicks and Mrs. B. T. Whitaker. Mrs. Whitaker's beautiful floral arrangements decorated the tables.

District X

The doctors' wives of District Ten were invited to a luncheon meeting at the Osceola Country

Club on September 21. Mrs. E. E. Lauvstad, of Osceola, served as hostess.

Although the attendance was small, the meeting seemed very worthwhile. Mrs. B. F. Kilgore, the state president, Mrs. E. A. Vorisek, the state finance chairman, Mrs. C. A. Trueblood, the councilor for District Ten, and Mrs. Hazel Lammey presented the various phases of the work of the Auxiliary.

Renewals of membership from two members were received, and two new members were welcomed. These members also contributed to the Health Educational Loan Fund and the AMEF.

Interest was shown in the legislative presentation, in Milestones to Marriage, in Future Nurses Club activities, and in the essay contest. An improved understanding of the various projects and problems of the Auxiliary assured the support of those in attendance.

District II

The members of the Woman's Auxiliary in District II attended a meeting and a following luncheon at the Green Mill Restaurant in Mason City on Tuesday, September 26. The Cerro Gordo County Auxiliary members were the hostesses, with Mrs. John B. Dixon and Mrs. Paul Potter greeting the guests, and Mrs. Stanley Vegors taking charge of registration. The following state officers and committee chairmen reported, each on her own phase

A group of members and guests at the District II meeting in Mason City on September 26. In front are Mrs. J. B. Dixon, president of the Cerro Gordo County Auxiliary, and Mrs. G. I. Tice, councilor for District II. In back are Mrs. B. F. Kilgore, president of the state Auxiliary, and Mrs. S. S. Westly, of Manly, a past-president.

of the Auxiliary's program: Mrs. E. B. Dawson, Fort Dodge, president-elect; Mrs. R. E. Hines, Des Moines, by-laws chairman; Mrs. E. A. Vorisek, Des Moines, finance chairman; Mrs. S. P. Leinbach, Belmond, Rural Health chairman; Mrs. R. F. Nielsen, Cedar Falls, immediate past-president; and Mrs. Hazel Lammey, Des Moines, administrative secretary. Since she was Civil Defense chairman last year, Mrs. Leinbach reported on that program, in the absence of the current chairman, Mrs. Sidney Brody, of Ottumwa. Mrs. T. E. Davidson, of Mason City, reported on the legislative program being carried out in Cerro Gordo County. The AMEF project, publicity and publications, community service, handicapped craft sales, health careers, the Health Educational Loan Fund and statewide participation in the legislative program were also discussed by the officers.

The presidents of Auxiliaries in the three organized counties in the district gave reports, outlining their groups' participation in the programs, and members-at-large from Hancock County reported on their participation and cooperation in community projects. Mrs. S. S. Westly, of Manly, was introduced. She is a past-president of the state Auxiliary and an active member of the Worth County organization.

At the luncheon, Dr. Jay E. Houlahan, a member of the Iowa Medical Society's Advisory Committee to the Woman's Auxiliary, and Mr. Doug Sherwin, of KGLO and KGLO-TV, Mason City, were guests. Speaking to the group, Dr. Houlahan emphasized the importance of the pending legislation concerning medical care and the necessity for each individual's being well informed about it. He also related what is being done in District II in this regard, through meetings with non-medical groups. He suggested working out essay contests at the local level to emphasize what is happening in the socialization of our economic system. Mr. Sherwin discussed the program "Ask the Doctor" which is currently being televised by KGLO-TV on Fridays from 4:30 to 5:00 p.m. He commended the Cerro Gordo County Medical Society and the individual doctors for their cooperation in making the broadcasts possible.

A brief question period followed. One of the suggestions that arose from that part of the program concerned making the study of the social security system a part of high-school social-studies classwork. The proposal to include medical care benefits under social security would, of course, be examined carefully as a part of that work.

IN MEMORY

Mrs. Emory Mauritz, Des Moines, August 26, 1961. "Our path emerges for a while, then closes with a dream."

—ERNEST DOWSON

COUNTY AUXILIARIES

Mahaska

The Mahaska County Medical Auxiliary met for a one o'clock luncheon at the Downing Hotel, in Oskaloosa, on Tuesday, September 19. Seven members and two guests were present.

Mrs. L. F. Catterson, councilor for District IX, outlined plans for a district meeting scheduled for October 18, at the Maple Room, Knoxville. A tea for the Future Nurses Club, to be held October 31 with the Mahaska County Nurses Association as co-hostesses was discussed. The Auxiliary undertook to provide decorations and furnish cookies.

Wapello

The Wapello County Medical Auxiliary held its first fall meeting October 3 at the home of Mrs. Loren Coppoc. Twenty-seven members were present. Earlier meetings included a morning coffee, for convention reports, at the home of the president, Mrs. Nelson Melampy, and a June picnic with the doctors at Loch Burn.

The Wapello County Auxiliary sponsors a student nurse in training and the Future Nurses Club. The members also voted a donation to the American Nurses' Foundation and one to the Community Chest. Volunteers will help with the Christmas Seal campaign.

Dr. P. D. McIntosh gave a most informative and provocative talk on fall-out shelters. He had recently returned from the Office of Civil Defense, in Battle Creek, Michigan, where the planning engineers have their headquarters. He feels that the Auxiliary should play an important role in alerting the public to the work each person must do in preparing for survival. His own neighborhood group is seriously planning the erection of a practical shelter. The Auxiliary members were all aware that tension had increased considerably since they held their last year's meeting for a discussion of civil defense.

A social hour followed, with Mrs. Dale Emerson and Mrs. William Whitehouse assisting.

JOURNAL of The IOWA MEDICAL SOCIETY

IN THIS ISSUE:

DECEMBER, 1961

when urinary tract infections present a therapeutic challenge...

CHLOROMYCETIN®

(chloramphenicol, Parke-Davis)

Often recurrent... often resistant to treatment, urinary tract infections are among the most frequent and troublesome types of infections seen in clinical practice.[1,2] In such infections, successful therapy is usually dependent on identification and susceptibility testing of invading organisms, administration of appropriate antibacterial agents, and correction of obstruction or other underlying pathology.

Of these agents, one author reports: "Chloramphenicol still has the widest and most effective activity range against infections of the urinary tract. It is particularly useful against the coliform group, certain Proteus species, the micrococci and the enterococci."[1] CHLOROMYCETIN is of particular value in the management of urinary tract infections caused by *Escherichia coli* and *Aerobacter aerogenes*.[3] In addition to these clinical findings, the wide antibacterial range of CHLOROMYCETIN continues to be confirmed by recent *in vitro* studies.[4-6]

CHLOROMYCETIN (chloramphenicol, Parke-Davis) is available in various forms, including Kapseals® of 250 mg., in bottles of 16 and 100. See package insert for details of administration and dosage.

Warning: Serious and even fatal blood dyscrasias (aplastic anemia, hypoplastic anemia, thrombocytopenia, granulocytopenia) are known to occur after the administration of chloramphenicol. Blood dyscrasias have occurred after both short-term and prolonged therapy with this drug. Bearing in mind the possibility that such reactions may occur, chloramphenicol should be used only for serious infections caused by organisms which are susceptible to its antibacterial effects. Chloramphenicol should not be used when other less potentially dangerous agents will be effective, or in the treatment of trivial infections, such as colds, influenza, or viral infections of the throat, or as a prophylactic agent. *Precautions:* It is essential that adequate blood studies be made during treatment with the drug. While blood studies may detect early peripheral blood changes, such as leukopenia or granulocytopenia, before they become irreversible, such studies cannot be relied upon to detect bone marrow depression prior to development of aplastic anemia.

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The Driver and Legislation

WILLIAM F. BURNETT

DES MOINES

Traffic officials know that the major factors affecting our traffic fatality rate are (1) excessively high speeds for existing conditions, (2) improper driving attitudes and (3) poor driving habits. But which conditions are the ones that need changing, so that drivers will have enough of a safety margin to save their lives? And how can driving attitudes and habits be sufficiently improved? We lack definite answers to those questions. Just as a disease must be pinpointed before it can be cured, we need to narrow our diagnosis and devise laws and programs that are precisely suited to our needs.

For a number of years, the American Cancer Society has stressed education and research as its two objectives. Those lines of endeavor are just as appropriate in our efforts to prevent traffic accidents as they are in that Society's attempts to curb deaths from cancer. At present, however, Iowa's traffic research is almost at a standstill because of a lack of funds, and the Safety Education Division hasn't money with which to undertake a mass safety education program in this state.

IMPROVEMENTS FOR ROADS, DRIVERS AND CARS

Only with research can we pinpoint our individual state's problems, and without it we spin the bottle and hope to find the right answer. Here are some of the questions that I, personally, would like answered. Why does Iowa have such a high secondary-road death rate? Those roads accounted for only 17 per cent of our travel in 1960, yet 40 per cent of our fatalities in that year occurred on them. Thus, proportionally, there were almost 2½ times as many deaths on secondary roads as there were on primary highways.

"Why?" we ask ourselves. "Why, mile for mile of driving, are there in excess of twice more fatalities on subsidiary roads than on main traffic arteries?" Could the smallness of our highway patrol be the reason? Our officers cannot possibly patrol the lesser roads frequently enough to enforce our laws there. Many of the deaths can be attributed to high speed on gravel roads. We do know that a majority of them involve just one vehicle and are listed as due to loss of control.

Could the concentration of fatalities on secondary highways be due to the way in which they were built? As most of us know, these secondary black-top roads have very steep ditches and have no shoulder areas to give the heedless driver a margin for error. This could be a problem point. Or possibly the driver is lulled into a false sense of security by the virtual absence of traffic deep in the country. We think we know the answers to some of the many and varied traffic problems in our state, but of course we should like to pinpoint them for work efficiency. We should like to give our "patient" a more thorough examination, but we lack a complete set of tools.

Once the problem has been discerned, an objective can be set. The Department's objective has always been, and will be, the safety of every individual Iowa citizen. Many times, the attainment of an objective involves legislation, for without proper laws, your enforcement body is hamstrung.

Both long- and short-term plans must be laid. For example, driver education courses are considered a long-term approach to the solution of our traffic problem, and increasing the number of patrolmen would be a short-term approach. Iowa has more primary and secondary road mileage in proportion to its area than has any other state. I should like to point up this fact for you. In Connecticut, the state with the lowest traffic death rate, there were 2.6 deaths per hundred million miles driven in 1958, as against Iowa's rate of 5.6 deaths per hundred million miles driven. Why this great difference? Connecticut had 400 patrolmen as against Iowa's 300, but the really significant fact is that Connecticut has a total of just 15,000 miles of primary and secondary roads, whereas Iowa has 109,000 miles. If the number of patrolmen on the Connecticut force in 1958 were considered ideal, then Iowa had no more than one tenth of the proper number for the job they were expected to do. Increased enforcement is not the entire answer to the problem, but it is an important segment of the over-all solution.

How should we treat our traffic ailment? I suggest a three-fold approach. Improving our drivers should be one phase. The Iowa driver needs to be better trained in driving methods. This might be accomplished in a number of ways: (1) A strong bicycle-safety program should be instituted, since the bicyclist is our beginning driver. (2) A thorough and complete driver-education program should be set up in our high schools, both public and private. (3) A stronger driver licensing program should be devised and instituted. There are many Iowa drivers on our roads today who have

Mr. Burnett, director of the Safety Education Division of the Iowa State Department of Public Safety, gave this address at the 1961 Annual Meeting of the Iowa Medical Society.

never taken a written exam on our traffic laws, and have never passed a practical driving test.

Setting up a more demanding examination for drivers might help. Today, the requirements aren't equal throughout the state. A citizen of Des Moines has to pass a more arduous practical driving test than does a citizen in a smaller community, mainly because there is heavier traffic at the test site. We might establish driver-improvement schools, designed to reeducate poor drivers. New York City has such a program.

The second phase of our program should be aimed at making automobiles as safe as possible for the people who ride in them. Great strides have been made in this area. The safety features available on our modern vehicles include the safety door latch, the recessed steering wheel, the twin-beam headlight, the padded instrument panel and visors, enlarged tail lights, reflectorized license plates, windshield washers, seat belts and many others.

Iowa presently has a massive voluntary vehicle-check program in operation. In 1958 there were 25 communities participating, and this year we expect that 200 communities will have participated.

The third approach to better safety is, of course, improving our highways. All curbing could be eliminated. The death rate on Iowa's interstate system was around 2.5 per hundred million miles driven in 1960. The death rate on our primary and secondary roads was 4.3 deaths per hundred million miles driven. Thus, quite obviously, good roads make a great difference. The driver, the car and the roads are the general areas for improvement.

LEGISLATION

During the legislative session this year, two safety bills were introduced, one of them the so-called "implied consent" proposal and the other a driver education bill. The implied consent bill would enable enforcement officials to file indisputable information against drinking drivers. I shall quote part of an editorial from the March 18, 1961, issue of the DES MOINES REGISTER regarding it: "We cannot understand the opposition that exists to the bill for checking up on drunk drivers. The proposed law would violate no one's constitutional rights. It would not force anyone to submit to a scientific test." Many opponents of this proposal have cited the United States Constitution and the Fifth Amendment against self-incrimination, and have claimed that an implied consent law would infringe on that right. The State of Iowa has no constitutional privilege against self-incrimination, but New York State does, and the Court of Appeals there (New York's supreme court) has held that a chemical test law does not infringe on a citizen's rights.

In 1959, the United States Department of Commerce drafted a report entitled "The Federal Role in Highway Safety." On page 25 of that report there is a discussion concerning how many drinks, on the average, a person would have to consume before reaching a blood alcohol concentration above .15 per cent: "One study which measured the concentration of alcohol showed that about 50 per cent of the drivers who were killed in collisions involving only their own vehicle, had blood alcohol concentrations above .15 per cent. This alcohol concentration is roughly equivalent to the consumption of approximately eight drinks in one hour by an average person." The State of Iowa is badly in need of implied consent legislation.

Another important piece of legislation is the Driver Education Bill. A number of studies have shown that approved courses in driver education reduce accidents. In a study of automobile fatalities among 16- and 17-year-olds that occurred during 1960 in Iowa, the Safety Education and Statistical Divisions of the State Department of Public Safety found that of the 53 drivers involved in death-dealing accidents, only one had received driver education.

The Iowa Department of Public Instruction reports that 59.5 per cent of eligible students in the 16-17 year age group receive driver education. This probably is a greater percentage than that for any other non-required school subject, but it isn't yet high enough. Virtually every one of those youngsters will shortly be driving a motor vehicle, and thus virtually every one of them should be trained to drive carefully, knowledgeably and responsibly.

THE PHYSICIAN'S ROLE

What does your Department of Public Safety need from you physicians? It needs your individual and non-official support for its programs. An informed public assures good legislation. Gentlemen, as individuals at the community level, you can and you should tell people what Iowa needs if it is to achieve a greater degree of highway safety. The people, then, will support the passage of needed laws and will endorse greater financial support for the Department's research, education and enforcement programs.

"It's unwise to pay too much, but it's worse to pay too little. When you pay too much, you lose a little money—that is all. When you pay too little, you sometimes lose everything, because the thing you bought was incapable of doing the thing it was bought to do. The common law of business balance prohibits paying a little and getting a lot —it can't be done. If you deal with the lowest bidder, it is well to add something for the risk you run, and if you do that you will have enough to pay for something better."

—JOHN RUSKIN (1819-1900)

The Crash: A Panel Discussion

Built-In Car-Crash Protection: A Design for Living

HORACE E. CAMPBELL, M.D.
DENVER, COLORADO

RESEARCH ENGINEERS at the Institute of Transportation and Traffic Engineering of the University of California at Los Angeles have estimated that a half of all the cars that are made will be involved, sooner or later, in injury-producing crashes.[15] A similar conclusion can be reached as follows. Something like 6,500,000 new cars are put on the road each year. On the basis of a very careful house-to-house canvass, the United States Public Health Service estimates that some 5,000,000 persons each year in this country are disabled in car crashes for lengths of time beyond the day of the mishap. Based on an average car occupancy of 1.7 persons, one can then say that there are about 3,000,000 injury-producing crashes per year—about half as many accidents as there are new cars in any one year.

Commenting on such estimates as these, an editorial writer for the NEW YORK TIMES said, as long ago as January 7, 1955, "Thus it would seem incumbent upon automobile manufacturers to view every car coming off the assembly line as though it may be involved in a crash, and to design it with that assumption in mind. Preoccupation with the design of hoods and tail lights should give way to a greater concern with the center and interior of the car, where, after all, its occupants are."[8]

A full-page editorial in the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION for June 11, 1955, entitled "Wanted: Safety Devices for Automobile Passengers," pointed out the design features that were needed to assure crash safety for the car occupants.[9] In the July, 1955, edition of TODAY'S HEALTH, another AMA publication, Vivian Rodell urged "Let's Build Safer Automobiles." She pointed out those features which were responsible for the deaths and injuries, quite apart from whatever may have caused the "accident." And then, in the fall of 1955, appeared the 1956 models. They possessed two new safety features, deep-dish steering wheels and safety door locks, as standard equipment, and crash padding and seat belts as optional equipment. All of the manufacturers stated that these were but a first step, and one car company promised many added features in the next year's model.

In February, 1957, after a year's use of the 1956 models and a good look at the 1957 ones, Paul W. Kearney contributed a most significant article to HARPER'S MAGAZINE, which I hope many of those interested will read or read again. It was entitled "How Safe Are the New Cars?"[13] His answer was that they were *not* safe, and he suggested a way in which they could be made safe. He wrote: "No industry has done a finer job in promoting safety and reliability in its field than the American Gas Association, whose members produce products with a lethal potential at least as great as the automobile. But, by establishing rigorous safety standards, and granting the coveted AGA label only to gas appliances which meet those stand-

Dr. Campbell is chairman of the Automotive Safety Committee of the Colorado Medical Society, a member of the Colorado Citizens Traffic Safety Committee, and a member of the Committee on Alcohol and Drugs of the National Safety Council. He and the authors of the three following papers made their presentations at the 1961 Annual Meeting of the Iowa Medical Society.

ards, this industry has commendably compelled its designers to think of safety first and styling second.

"In the realm of electrical appliances and fire protection devices, the pioneer UL symbol of the Underwriters' Laboratories has stood in the same enviable position for an even longer time. Here many different industries participate in paying the costs of brutal, impartial testing by an organization of hard-boiled experts, sponsored by the one group with the most to gain from safety: the insurance companies."

Then he goes on to say: "Nobody has ever questioned the fact that the engineers know as much or more about safe car design than any of their critics. *But they will never be allowed to put that knowledge fully to work until the sales executives are banned from the drafting rooms, just as they've been banned in the field of washing machines, electric blankets, gas furnaces, oil burners and a score of other devices with high accident potentials."**

I was privileged to participate in a panel discussion of crash safety at a meeting of the American College of Surgeons at Atlantic City in November, 1954. On the twenty-third of that month, the late Dr. Claire Straith, of Detroit, a plastic surgeon, wrote to me: "For over 20 years I have been trying to get the automobile industry to reduce the hazards by correcting some of the features about the interior design of their cars, but I find that it has been a very uphill business.

"It has been my experience after talking many times at their request, to the Automotive Engineers and the Society of Interior Body Designers and others, that although the designers themselves know of these hazards, the sales department is the one that throws up their hands if any mention is made of a possibility of their beautiful car being involved in an accident, and therefore they have been very hesitant to remove some of the shiny gadgets from the dashboard and to crashpad the same."

After all these years, crash padding is not standard equipment, and even the optional padding is not very good. But more of this later.

Beginning as early as 1934, Dr. Straith and Dr. E. S. Gurdjian, a neurosurgeon, both of whom—interestingly enough—were from Detroit, had pointed out that the injuries in car crashes were influenced by specific design features of the car interiors, and had made specific suggestions as to the modification of those features.[10, 14, 23-25]

Perhaps the definitive paper in this field is one by Dr. Fletcher D. Woodward, in the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION for October 30, 1948. In some way or other, this paper should be presented to the medical profession and the motorcar industry every year, until the suggestions in it have been adopted and used. But the vehicle safety engineer of our largest company said to me, "If you keep on this track that you are headed on now, you will become as big a crackpot as that fellow Woodward." But now, seven years later, I am still struggling to attain that distinction.[26]

One more reference to the efforts by the medical profession should be cited. The leading article in the November 5, 1955, JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION was written by Dr. C. Hunter Shelden, another neurosurgeon, and was entitled "Prevention: The Only Cure for Head Injuries Resulting From Automobile Accidents." It should be pointed out that its trenchant tone stems from a visit that the author had made to the car manufacturers in Detroit and from his discovery of their attitudes toward crash-safety design. He said, "The reason is that the industry is apparently governed by the cost accounting division."[22]

CAR MAKERS HAVE DRAGGED THEIR FEET

Now it may be suggested by defendants of the industry that this is all "very old stuff," and that there has been steady improvement in crash-safety design since the advent of the 1956 models. But listen to Robert L. Candlish, the Detroit editor of DESIGN NEWS, an engineering journal of fascinating interest, and himself an engineer of 20 years' service in the motorcar industry. He wrote just this spring, in an editorial entitled "Stagnant Progress": "Among the devices credited to the generosity of the industry are four-wheel brakes, safety-glass windshields, turn signals and windshield washers. Four-wheel brakes were adopted in the mid-twenties, and safety glass was required by law in the early thirties. Turn signals also become standard fittings when required by law in many states. Where are examples of more recent progress in safety devices? . . .

"The automotive industry's interpretation is that design in regard to safety has gone as far as it needs to go. Actually, the high point in design safety was reached some time ago. When considering *standard car models,* there was without doubt as much safety in the car of 25 years ago as there is in those built today. . . ." He continues with details and examples, and then concludes: "A retreat from reality is poor design."

The editor of DESIGN NEWS, Mr. Ernest R. Cunningham, wrote a leading editorial for the November 7, 1960, issue entitled "Auto Safety—A Record." In it, he said: "Space will be made available to any senior automotive executive responsible for policy decisions on safety who feels he can explain or justify the automotive industry's record on safety—or the design from the standpoint of safety of his own company's new models."[5] As this is written, five months after that announcement, no one has accepted Mr. Cunningham's offer.

Safety glass is often ballyhooed as a contribution

* The italics are mine.

by the industry. After thousands of cases of horrible lacerations, we finally got safety glass of sorts —after a very expensive law suit and specific legislation.

A manufacturer who has done as much as any other to prevent crashes has made available a sheet which is reproduced herewith as Figure 1. It appeared in the National Safety Council's publication, TRAFFIC SAFETY, in August, 1958.

Let us check off the items shown there. It has been stated that it takes two years for an item of design to get on the road, so we might have expected the 1961 models to be equipped with some of the improvements mentioned in Figure 1. Yet I know of only one car that has even one of these suggested items!

SAFETY STEERING WHEELS

Let's start at the top of the page and consider the safety steering wheel. The wheel depicted is better than any now used, and I ask why the Society of Automotive Engineers hasn't yet arrived at a good standard non-lethal steering wheel. I think the Insurance Institute for Highway Safety should insist upon the development of the best possible device of this sort. The steering wheel kills and injures more people than any other single item in the car.

The present Ford wheel has energy-absorbing spokes, but the force is applied by the wheel rim to the upper abdomen when a crash occurs. Chest injuries will certainly be diminished by this wheel, but upperabdominal and face and laryngeal injuries are being increased by this type of wheel. The two-spoke wheel used by other car makers—the spokes forming a sort of broad yoke against which the chest is to be impacted—would seem to provide better protection IF the wheel, at the moment of impact, is in straight-ahead rotation. But if the wheel is a quarter-turn off center, it is worse than the old one. The wheel depicted in Figure 1, with the circular energy-absorbing pad, is soundly designed, but show me one on an American car! The Mercedes Benz has one, but this is the year 1961, five years after the first safety steering wheel came out, and the development promised in the American manufacturers' publicity at that time is still just publicity.

DASHBOARD IMPROVEMENTS

Look now at the right side of the dashboard that is pictured, and notice the increased jackknifing clearance provided by the suggested flattened and padded panel, and note the divided front seat, the righthand section of which is anchored securely to the floor so far back that a belted passenger could swing forward over his belt and strike nothing. This is a most significant design advance, but have you seen a car designed this way? I know of three makers that have made a short step in this direction, but most of the cars have a deep shelf with a sharp edge pointing right at you. The 1961 Chevrolet, in my opinion, is the worst of all in this regard. As a matter of fact the designs for the 1950 and 1951 cars of that make were better in so far as dashboard design was concerned. Why the glove compartment has to have a sharp shelf over it is something that is hidden in the murky depths of some stylist's mind. Such surfaces, furthermore, should be formed of ductile metal, whether subsequently padded or not. One wonders whether the vehicle safety engineer in that organization had anything at all to say about those features—or indeed whether such men have anything to say about the "safety features" of cars coming from that particular maker.

It is a standard rule that the surfaces that may be struck by the right-seat passenger should all have curvatures with a radius greater than two inches, and two inches is approximately the radius of a softball. Just look at any of the instrument panels offered, and see which one meets this criterion and which one you want your passenger to strike at your next fast-brake stop.

Until the motorists can be persuaded to use belts and shoulder straps every time they get into a car, the least that car makers can do is to provide, as standard equipment particularly in models expected to have wide sales, forward surfaces that will minimize injuries as much as humanly possible. In my opinion, however, this will not occur until legislation requires it, either at the state or at the federal level. I think the time has come to have federal laws which will do for cars what the Pure Food and Drug Act, the Mine Safety Act, the Meat Inspection Act and other laws have done in their respective fields.

As I have indicated, it is my opinion that the basic design of the instrument panel on the American car of widest distribution is the worst ever—less safe by far than the instrument panel of the 1950 and 1951 models of the same make. One of the salesmen has told me that the optional padding is so scanty both as to depth and area that it is regarded by his sales force as a joke.

I present a sample of Ensolite, an energy-absorbing material made by the United States Rubber Company, of Mishawaka, Indiana. There may be other materials just as good, and rather than plugging a particular commercial product, I'm just showing you one of the materials that are available. We put a sheet of this material just one inch thick on the marble floor of the State Capitol rotunda in Denver. Then we dropped fresh hens' eggs from the topmost balcony above, from a height of 115 feet, down onto this rug of Ensolite. The four that hit fairly did not break. Two inches of material with the attributes of Ensolite should be standard equipment on all forward areas—i.e., instrument panel, windshield header, corner posts, side posts and at least the front half of the roof.

SAFETY FOR TOMORROW'S CARS

How can safety be built into the cars of the future? Ford engineers have come up with an experimental car that explores some interesting possibilities.

A SAFETY engineer's "dream car," equipped from rear seat to front bumper with devices designed to increase the chance of survival in the event of collision or rollover, has been developed by the Ford Motor Company.

The safety car, created on an Edsel chassis, is equipped with an energy absorbing front bumper to minimize the speed of deceleration in case of a crash.

The steering column hub, 11 inches in diameter, is made of foam plastic to protect the driver's chest in the event he is thrown forward with sufficient force to break off the steering wheel.

Both front and rear seats are designed to support the passengers' heads against whiplash during a severe rear-end collision. Foam cushion padding along the top and back of the front seats is designed to protect rear seat passengers who might be thrown forward.

All instruments on the dashboard panel are recessed and surrounded by foam plastic padding. Panel padding on the front seat passengers' side extends completely over the dash and almost to the floor. The glove compartment, which is normally on the right side of the instrument panel, has been moved to an area beneath the right front seat.

The front seat is a two-piece sectional, with only the driver's one-third section being adjustable, while the other two-thirds is fixed solidly to the floor.

Floor-anchored seat belts are on retractable reels equipped with automatic inertia-locking devices which are activated by sudden thrust.

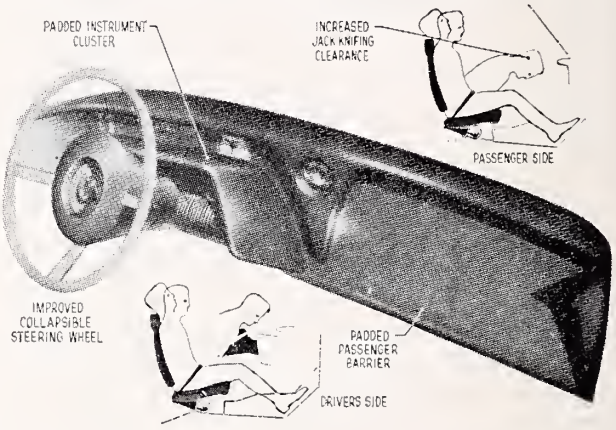
Door handles are built into padded arm rests and the window cranks retract into padded "buttons."

The roof and door pillars are padded and each door is equipped with warning lights that flick on when a door is open.

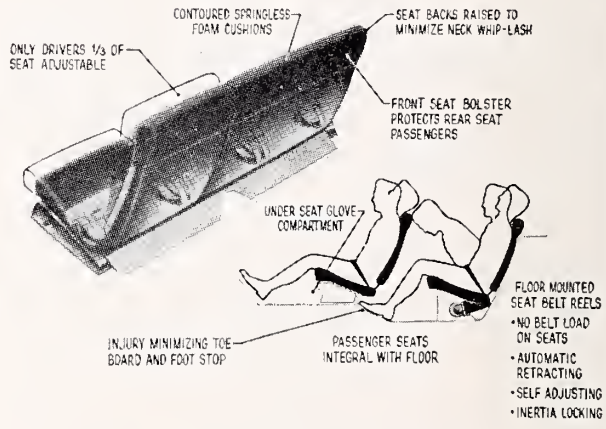
Evaluation of the safety car is part of Ford's continuing research program on automotive safety.

All features of the car are of experimental design and in various stages of development. While Ford engineers concede that some of the features may never prove to be effective or feasible, they point out that this type of investigative program has produced safety features already introduced by the company. They add that new advances will be forthcoming to further increase future driving safety.

SAFETY STEERING WHEEL AND INSTRUMENT PANEL



SAFETY SEATS



CRASH-ABSORBENT FRONT STRUCTURE

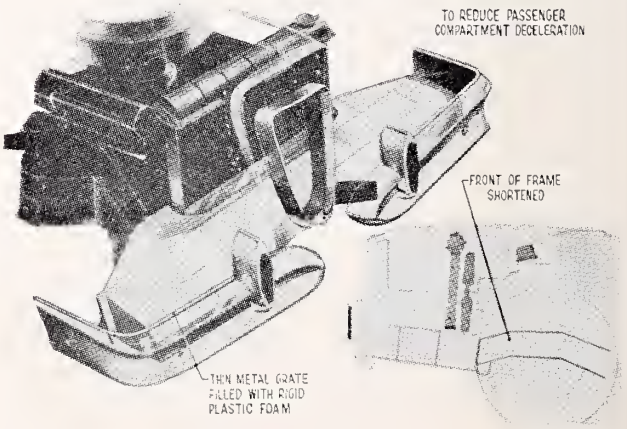


Figure 1. Safety features that were developed by Ford as long ago as 1958. Few of them have since been offered as standard equipment.

A prominent engineer, long a student of the decelerative problem, has worked out a device to hold a roll of this material above the windshield on the right side of the car, to be released by an inertia switch to drop down over the windshield on the passenger's side at the moment of impact.

I know of no padded instrument panel at present that has padding adequate either in thickness or in quality for the job that it may be called upon to perform. Reliable information has come to me that the purchasing agent for one of the larger companies refused to buy the better-quality material, and when told that the material he selected would not last very long, replied that if it lasted 90 days, it would be all right.

I was instrumental in obtaining new and better padding as a no-cost replacement for two doctors whose instrument panel padding had become hard and lumpy in something over a year following their purchase of new cars. They had paid for padding as an extra-cost feature.

Ensolite is a closed-cell plastic foam. It is not rubber and it is not a sponge. Each cell is separate from every other one, and thus the material does not absorb moisture. A piece has been floating in a pan of water for seven years, is weighed periodically, and has absorbed no water. It makes excellent athletic padding, incidentally. It is very little affected by light, and pieces older than seven years are still resilient and efficient. It will last as long as the car in which it is installed.

Let's move down to the center illustration in Figure 1. All of the characteristics of these seats are good, but the definitive point is that only the driver's section is adjustable, and the right two-thirds is integral with the floor. This is a terrific idea. Many motorists have been crushed by the seat they had been sitting on. This feature should have been in all cars years ago.

SEAT BELTS AND INERTIA REELS

In the smaller sketches below and to the right of the large one at the center of the page, are depicted the dream belts equipped with inertia reels. Don't try to buy one; you can't. Yet I saw them on the Ford X100 almost 10 years ago. I have roof-mounted inertia reels on the shoulder straps of my sedan. They are a standard military item, found on all military fighter planes, and I am confident that arrangements could be made to equip motorcars with them if crash-safety were a compelling item of interest for motorcar manufacturers.

But let's forget these dream items for a moment, and consider what has been done about the simple garden variety of seat belts. For years it has been extremely difficult to install belts in some makes of cars, and year after year it has become more difficult. It seemed as if the maker were trying to thwart the installation of belts. But now, we learn, this is over, and all manufacturers are going to make it easy to put belts into their cars. How did this change come about? Suffice it to say that the industry got busy on combustion-fume control, under the lash of California legislation. I think it is more than coincidental that the announcement of this seat belt improvement followed closely upon a proposal in the New York legislature for compulsory provision of seat-belt-attachment devices and a visit to Detroit by a joint committee of the New York legislature under the leadership of Senator Edward J. Speno.

CRASH-ABSORBENT FRONT STRUCTURES

Let us turn now to the bottom illustration, labelled "Crash-Absorbent Front Structure." There we find a publicized suggestion by Ford engineers "to reduce passenger compartment deceleration." This is of particular interest to me personally, as you will see.

At the American College of Surgeons meeting at Atlantic City in 1954, to which I have previously referred, I postulated as a necessary corollary of the seat belt, some type of energy-absorbing nose for the automobile. Such a device should absorb as much of the crash energy as possible and reduce as much as possible the crash load imposed upon the belt—i.e., allow it to remain intact at as high an impact as could be managed. Primarily, of course, the whole idea was to reduce to the lowest possible figure the crash load imposed upon the human organism.

As cars are now made, the bumper is nearly worthless as an energy absorbing mechanism. Furthermore, the fenders and your ever-so-expensive and lovely grille are no better, though the radiator and water pump are fairly efficient. The result is that the frame structure and engine block are called upon to do most of the energy absorbing. In consequence, the degree of deceleration is very high, there is extensive car damage (part of the planned package), and most serious of all, a terrific load put upon seat belts even during relatively low-speed impacts. You will hear that the generous overhang at the front end of your recent model is an efficient energy shock-absorber. Don't believe it. This is a contrived myth.

This has been shown up as a fallacy by a carefully documented experimental crash at the Institute of Transporation and Traffic Engineering, in which a car impacting a heavy concrete barrier at only 28 mph. suffered a frame deceleration of 59 G, with seat belt loads of 34 G. In other words, a 200-pound man would have broken his SAE belt at 15 G, well before the peak of the crash. Even the GSA 5,000 lb. belt would have parted at 25 G, well after the SAE belt, but uncomfortably far from the peak. Only the 8,000 lb. Air Force harness would have kept the occupants in their seats, and this, mind you, at only 28 mph. And even this wouldn't have been too relevant, for the passenger space was seriously encroached upon when the front structures came back into the front pas-

senger compartment. The mean effective stopping distance in this crash was only 5.3 inches.

Consider now what the circumstances would have been if two full feet of uniform deceleration had been provided anterior to the frame elements of the car. This would have lengthened the car very little, and would merely have put to good use the two feet of chrome ornamentation that now occupies the front areas of our cars.

The frame deceleration would have been somewhere in the neighborhood of 15 G, the seat belt load would have been somewhat less, and even the old SAE belt, with its standard of 3,000 lbs., would have held a 200-pound motorist in his seat. The whole character of the crash would have changed from a destructive and lethal calamity of the "bridge-abutment" variety to one of no serious import either for passengers or car, for if the motor did any damage it would have been to the frame member in front of the motor, if its attachments to the frame were of insufficient strength.

At that Atlantic City meeting, I suggested that the energy-absorbing material might well be aluminum foam, a light material requiring no maintenance, which could be removed after the crash by means of a wrench and the release of three or four bolts. This idea was greeted with derision by a certain segment of the motorcar industry, and the remark was made that no one had ever seen aluminum foam. I had done a little work with a piece of aluminum that had foamed accidentally when I was a student in college physics, but to be sure, I had never seen a piece of the commercial stuff. Well, here is a block of aluminum foam, and I can inform the automobile industry that its makers are ready and willing to provide unlimited quantities of it for their cars.

Here is another material which has phenomenal energy-absorbing qualities, Hexcel aluminum honeycomb. Its energy-absorbing capabilities are strictly along the axes of the cells, so it shouldn't be squeezed laterally. For this reason, it may not be so well suited for the front ends of cars as is aluminum foam, but here are two materials that the motorcar industry should in all conscience investigate.

Quite apart from these new materials, Figure 1 indicates the use of rigid plastic foam. Here is a piece of such material. It is Ensolite in a woodlike form, and it can be made even firmer for high energy absorption. Also, it can be made soft enough for use in padding men's suits. And here is an even more recent development. It is doughy in consistency, and is the most efficient substance yet developed for use in the interiors of cars.

THE ATTITUDE OF A TYPICAL MANUFACTURER

The hydraulic bumper with a variable escape aperture is strongly to be considered. It is probably the most effective energy absorber of all, as witness the shock absorbers in use on all cars. A light, low-cost, "one-shot" hydraulic bumper has already shown what it can do.

Let us now investigate an official industry reaction to the energy-absorbing bumper. On Monday, August 27, 1956, before a hearing of the Roberts Committee of the House of Representatives, held in Detroit, Mr. Charles A. Chayne, vice president in charge of engineering at GM, testified in detail and *in extenso*.[4] The following ones of his statements appeared on page 359 of the transcript of the hearings: "Modern vehicle front ends comprise such components as fenders, fender innerskirts, radiator supports, grilles, other front-end sheet metal and, of course, the front bumpers. At various times the industry has been subjected to criticism of these front-end designs and for what is often termed an excessive amount of front-end overhang—that is the amount of front structure that is ahead of the transverse centerline of the front wheels and forward of the ends of the frame side rails. However, these designs, we think, have been vindicated as a realization has been gained of the important part they play in high crash-energy absorption."

A prominent research engineer, who has no vested interest in the motorcar industry, permits me to quote him as follows: "In my opinion, this comment on bumpers is completely absurd." Another prominent research engineer states: "Engineers know how to provide crash safety in automobiles. It is to their ever-increasing shame that they have not utilized that knowledge."[19]

The crushing of these front-end structures does indeed absorb a modicum of energy, but it is largely negligible compared to the forces generated, as the well-documented crash described above amply demonstrated. Every one of the manufacturers has staged similar crashes and knows precisely what a small precentage of the crash energy is absorbed by the front-end structures named. It is the frame and the engine block which absorb the energy, with great destruction to each and no protection to the passengers. I have documental evidence of injuries inflicted by the engine's coming back into the front passenger compartment.

Having attributed so much to the structures enumerated, all of which are expensive to replace, Mr. Chayne went on to ridicule the concept of the energy-absorbing bumper. He hypothecated a car traveling at 20 mph. and contacting a concrete culvert structure and stopping within one foot, with a resulting deceleration of 13.4 G. (The table that he used corresponds exactly with mine!) He thoughtfully stated: ". . . the bumper, fenders, grille and front-end sheet metal are pushed back or deformed one foot—which actually represents the stopping distance." Interestingly enough, he failed to mention the forward ends of the frame side rails. I should be willing to wager a very small sum that every car that his corporation has built with frame side rails will allow these to come into contact with a concrete barrier in a collision at 20 mph.

Then he went on to say, with simulated scientific

precision and impeccability, that a bumper to be worth anything at 20 mph. would have to extend in front of the car for 178 inches—a matter of 15 feet. One of the congressmen present commented that a car so equipped would require a very long garage. What the congressman did not detect was that Mr. Chayne had based his ridicule of the energy-absorbing bumper upon the premise that it was expected to reduce the impact suffered by the occupants to the level of that produced by a "panic" brake stop on dry concrete, in a car equipped with good brakes and plenty of tread on the tires. His calculations had been impeccable and his premise had been explicitly stated in a previous paragraph, but his premise was quite irrelevant. Professor Bruhn called it "absurd."

No one other than Mr. Chayne and Mr. Karl Richards has ever said that any real benefits could be expected from an energy-absorbing bumper, as far as car occupants are concerned, unless seat belts were used. Let me say as explicitly as I can that the need for a good energy-absorbing bumper is predicated strictly upon the use of some form of personnel stabilization—e.g., seat belts. And just as explicitly, it should be said that the full potential of seat belts will not be realized until they are accompanied by good energy absorbers at both the front and rear of the car. Let me hasten to add that seat belts have almost no value to the people whose car is struck from the rear.

It is no accident, I should guess, that the engineer who has ridiculed the energy-absorbing bumper is the same whose products, year after year, have had front seats designed in such a way as almost to preclude the installation of seat belts.

My further interest in the Ford Motor Company's proposal to "reduce passenger compartment deceleration" by means of a "crash absorbent front structure" is heightened still further by the fact that some of my colleagues in the American College of Surgeons have labelled my suggestions for an energy-absorbing nose for the automobile and for the use of aluminum foam for this purpose as "absurb" and "ridiculous" in various official minutes. And Mr. Chayne's statements have continued to be relied upon. In the April, 1958, issue of AMERICAN HIGHWAYS, nearly two years later, my co-panelist Mr. Karl Richards carried out the very same argument in an article entitled "Five Fallacies in Traffic Safety." He used the same illustrations and the same figures as those previously employed by Mr. Chayne, and wrote: "But the greatest gains in crash-injury prevention have come through postwar design and construction of the cars themselves—designs which can absorb crash energy in this way offer added protection to car occupants." He closed that section of his paper by saying, "Occasionally we hear people advocate special shock-absorbing bumpers for protection here."[17]

Now much as I admire the Ford Motor Company for what it has tried to do for crash safety in spite of the toughest kind of opposition, I must point out that its engineers have sketched this energy-absorbing structure as completely enclosed so that these self-same structures—the fenders, lights, fender inner-skirts, radiator supports, grilles, and even the water pump and fan—are sacrificed to the cause of safety. Of course, even Ford has to keep its dealers' body shops active and reordering spare parts!

CONCLUSION

I trust it will be recognized that I have endeavored to keep this discussion on a broad generic plane and have resorted to few personal references. In order to give you the realities of the situation, however, I can do no better than to quote from the CONGRESSIONAL RECORD for August 24, 1959, covering the debate on H.R. 1341, a bill introduced by Congressman Kenneth A. Roberts, of Alabama, seeking to require certain minimum safety devices approved by the Bureau of Standards in cars purchased for federal use.

Most of the opposition to this Bill came from two congressmen from Michigan, Hon. George Meader and Hon. John B. Bennett. The specific statement that I wish to quote is one by Mr. Roberts that appears in the first column on page 15440: ". . . Let me say this, that in the hearings industry was represented. We had a full hearing. They had all of their vice-presidents in charge of engineering to come into our hearings. And I could tell you as a member of this body that four out of five members of the automotive industry were willing to endorse this bill and so communicated that fact. But one objected, and that is the reason there is opposition to this bill today."[18] Suffice it to say that this bill passed the House by a vote of 255 to 125, with defeat of a motion to recommit.

Despite the excellent record of the gas and electric appliance manufacturers, there is no evidence that the motorcar industry is any different from the other industries that required federal legislation for their control. We have the Pure Food and Drug Act, the Meat Inspection Act, the Mine Safety Act, the Railroad Safety Act, the Marine Safety Act, the Civil Aeronautics Act and others. It is high time that we enacted a Motorcar Safety Act for our personal protection.

The Roberts Bill was defeated in the last Congress because the Senate Subcommittee for Surface Transportation didn't feel the necessity of meeting to discuss it. Thus, the motorcar industry isn't the only group to display social irresponsibility. The proposal will have to be reintroduced, and all doctors should work for its passage. It is not a complete answer, but it is a first step toward the answer, and the first step must always be taken if any destination is to be reached.

The eyes of 1,250,000 motorcar dead, in the United States alone, are upon the medical profession!

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The Automotive Crash Injury Research Program of Cornell University

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A RESEARCH PROGRAM of Cornell University continues in an attempt to determine the causes of injury to the occupants of cars involved in accidents. These studies are designed to assist safety engineers in their efforts to design cars so as to minimize the risk of injury if an accident should occur, and they supplement the commoner approach of seeking to determine the causes of accidents. It is, of course, to be hoped that accident prevention may someday become so effective as to make crash-injury research unnecessary, but in the meantime, with an estimated 4,700,000 persons injured annually in automobile accidents,[1] our approach is a necessary part of the overall program of traffic safety improvement.

THE SAMPLING

The Cornell program collects and analyzes information on motor-vehicle accidents that have taken place on country highways, rather than on ones that have been contrived and staged on automotive testing grounds. Data for ACIR research are contributed by state police organizations as a by-product of their investigations. Only injury-producing passenger-car accidents are reported to ACIR by these policemen, although for their own purposes they also investigate injury-producing smashes involving trucks or involving cars and pedestrians, and automobile accidents in which the consequences have been confined to property damage. When they have investigated a passenger car accident in which someone was injured, in addition to their own reports they fill out a special ACIR form that calls for details of the injury-producing elements in the crash. They also photograph the cars and submit pictures. The physicians who have treated the injured fill out other ACIR forms, giving details of the location, nature and severity of each injury.

About half of these accidents have involved forward force—i.e., the net result of all the forces has been such as to throw the occupants forward. In about 15 per cent of them, there have been left or right impacts, and rear-end collisions have made up the remainder. In about one out of five accidents there have been roll-overs, and in the other four out of five there have been some lateral forces.

A total of 21 states has contributed data to ACIR at one time or another during its existence. The 25,000 reports of injury-producing accidents that constitute a major part of the data were received as a result of extensive cooperation by participating organizations. The fact that these agencies have contributed so faithfully and have done so without compensation indicates their great interest in the problems of highway safety. ACIR is indebted to them.

ACIR data are collected according to an area sampling plan, when it is possible to make the necessary arrangements within the state. The plan is formulated in such a way as to guarantee that

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The author is the assistant director of the project that he describes.

an area from each geographical extreme will be sampled during each season of the year. Consistent with this restriction, areas are picked by a random probability method. That is, the probability of an area's being picked is proportional to the accident density within that area. But once an area is selected, it stays "active" for six months, and each injury-producing accident within its boundaries is reported.

Sometimes it is impossible to shift areas randomly, and therefore it is necessary to take what is available. Fortunately, even such samples as these seem adequately to represent the populations from which they are drawn. A study has shown that ACIR sample distributions of accidents by day of the week, age and sex of vehicle occupants, etc. were similar to population figures published by the states.[2] This was true for both types of samples. Another study[3] has shown that, despite differences among states and among samples, the basic relationships between accident variables and degrees of injury are quite consistent. These findings suggest the general applicability of ACIR results.

PROCESSING

Case materials are studied by ACIR analysts, coded and finally transferred to punched cards. Some items are recorded as matters of fact, but others involve complex and subjective judgments of injury severity, accident severity and extent of damage to the automobile. The importance of these subjective ratings is shown by the fact that injury severity is the dependent variable in most ACIR studies, and accident severity is the single most important control variable. A study has shown that these judgments have been made with a high inter-rater consistency. Disagreement among four judges has rarely been by more than one category. Moreover, no interaction has been found between ratings of two dimensions that are themselves correlated (e.g., accident and injury severities).[4]

FINDINGS

Ejection. One of the most fundamental findings by ACIR was published in 1958. At that time, the undesirable consequences of being thrown from a car during an accident were documented. It was shown that ejection occurred more frequently in accidents of greater severity, and it was determined that the frequency of fatal injuries among ejectees was more than twice as great as it was among non-ejectees. This finding countered the prevailing bit of folk lore that it is desirable to be thrown clear in a crash. This misconception had been perpetuated by occasional newspaper stories of someone's having been thrown clear of a wreck —onto a soft meadow, according to the typical story of this sort—and suffering no injuries at all.

In that same paper, it was estimated that 5,500 lives could be saved annually by the complete prevention of ejections.

Ejection occurs primarily as a result of doors' flying open during the crash sequence, and there are at least two approaches to the prevention of ejection. One involves restraining devices such as seat belts, which could prevent ejection even though doors open. The other involves door locks, which could reduce the probability of doors' opening. ACIR has conducted research in an attempt to evaluate both of those devices.

Seat Belts. Two ACIR papers concern the availability, use and effectiveness of seat belts in automobile accidents.[6, 7] Special data were obtained from California for these studies, and the first set dealt with the *usage* of seat belts. The study included data from 54,000 California accidents that occurred in 1958, involving a total of 94,000 vehicle occupants. It was found that a belt was available in each of 3,010 of the 94,000 occupied seats (3.2 per cent). Most of those belts were in front seats. It is unfortunate that so few seats were equipped with belts, and it is even more unfortunate that of the 3,010 occupied seats equipped with belts, only 977 (32.5 per cent) of the occupants were wearing their belts at the times of their accidents.

The benefits of seat belts to the 977 people who wore them at the times of their accidents was quite remarkable. Belt-wearers suffered 35 per cent fewer "major-fatal" injuries than did those who did not wear belts. Seat belts seemed associated with the greatest injury-reduction in rollover and side-impact accidents. These, incidentally, are the mishaps in which ejection occurs most frequently when occupants are not restrained. Thus, the belts seemed to have prevented ejection.

In summary, the probability of being injured to *any* degree was about the same, whether or not belts were worn, but there was a large, significant reduction in the *seriousness* of injury for those who were wearing belts. It appears that a major part of the advantage of belts is that they give occupants protection from the serious injuries that accompany ejection. However, it seems certain that belts don't necessarily prevent the occupant from being injured to *some* extent, a fact that of course isn't surprising. Belts meeting standards set by appropriate agencies are now widely available. Installation procedures have been formulated which capitalize on the unique structural strengths of various makes and models of cars. Beginning with the 1962 models, seat-belt anchors will be standard equipment for the front seats of passenger cars. Yet, there remains the problem of creating interest in the use of seat belts.

Door Locks. The second approach to the problem of ejection has to do with improving door locks. If ejection occurs because doors fly open, then it is logical that it could be prevented in most instances by keeping doors closed during crashes. For several years now, American automobile manufacturers, as well as some foreign ones, have equipped cars with improved door locks designed to prevent doors from opening during accidents. In 1956, ACIR made public the find-

ings from its preliminary analysis of door openings before and after the advent of the new type of locks.[8] At that time, it appeared that door openings, ejections and injuries were appreciably fewer in the newer cars. The sample had been small and the findings limited, but a thorough follow-up of those preliminary figures will be released soon. This new study will show that door openings have been reduced by about 35 per cent. Of course the figure varies above or below 35 per cent for the various categories of accident severity, body style, accident configuration, manufacturer, etc., but the overall reduction is gratifying indeed.

A unique advantage of preventing ejection through improving door locks is that the need for the vehicle occupant's cooperation is minimized. In order for seat belts to provide protection, they must have been installed and must be used. On the other hand, the door lock is available as standard equipment, and it operates without the necessity of any unusual effort on the part of the occupant. Yet even in the case of door locks, some degree of occupant cooperation is needed. Automotive safety engineers state that door openings would be reduced even further if occupants would keep their doors locked from the inside.

STUDIES IN PROGRESS

Some of the projects that are under way at ACIR include an evaluation of instrument-panel padding and/or recessed-hub steering wheels, a study of speed in injury-producing accidents, and a study of the differences between injury-producing and property-damage accidents.

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The Traffic Accident Enigma

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BY VIRTUE OF the development and use of the automobile, the United States pursues a pace that exceeds that of any other nation, past or present. That pace has a price-tag on it, which an outstanding economist called, some years ago, "the social overhead cost of our transportation system." He wasn't referring exclusively to deaths and injuries in our highway transport system, since we continue to have serious accidents on our railroads, in aviation and on water-borne ships, but he had our highways chiefly in mind. On the basis of the record over the past four decades, we must admit that the toll in lives, injuries and property destruction on our streets and highways vastly overshadows the combined toll from mishaps in other segments of our transportation system.

For more than 40 years, then, we have been trying to reduce or—for those who will settle for nothing less—to eliminate traffic accidents completely. The rational person would be satisfied with any substantial and lasting reduction.

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Except for the dedicated persistence of a very few researchers, during the period from 1920 to 1940 no sophisticated attention was given to man's performance in the system. Exactly when the change occurred is unimportant, but it is my impression that research into the human factors began in connection with military aviation and later was extended to civil aviation and then to surface traffic problems. I am referring to man-machine-environment studies.

It is difficult to identify milestones, at least in recent history, but momentarily throwing caution to the winds, I should say that the resolution from the Committee on Trauma to the Board of Regents of the American College of Surgeons, on February 5-6, 1955, was a milestone in the evolution of our philosophy regarding the reduction of highway casualties. In part, that resolution read as follows:

> BE IT RESOLVED that the Committee on Trauma of the American College of Surgeons requests that the Board of Regents of the College recommend to the motor car manufacturers of America that they stress occupant

safety as a basic factor in automobile design, to include (1) doors which will not open on impact; (2) seats and cushions which will not become displaced on impact; (3) energy absorbing interiors; (4) adequate safety belts and other passenger stabilizing devices that will resist impacts of at least 20 G.

Results have been slow in coming, for the simple reason that across-the-board changes in human habits aren't peremptorily ordered in a democracy. The automobile seat belt has been advocated for almost a decade, for example, but hasn't yet been generally adopted.

HIGH SPEEDS SEEM INEVITABLE

The head-on collision of automobiles is, perhaps, the most dramatic example of malfunctioning of our street and highway system of transportation. It provides the daily press with spectacular pictures, and coincidentally it lends itself to vivid description and emotion-arousing stimuli. For too long, such portrayals have been viewed as accident-prevention efforts. A variation of the horror-story technic has been the exhortation to reduce speed. Quite clearly, this has been and continues to be mostly wasted energy. Who are the greatest protagonists of speed? As evidenced by their actions rather than by their considered statements, they are none other than the members of the driving public. Speed of movement is one of the principal purposes of automobiles. Speed of movement for people and goods also happens to be one of the most important goals for our economy, or for the economy of any other country.

Studies by Moore and others have shown that approximately 80 per cent of all serious highway accidents occur at speeds of less than 40 mph. It should be clear, then, that if any substantial reduction in highway deaths and injuries is to occur through restrictions on speed, it will be necessary to change one of society's basic objectives.

EFFORTS TO PREVENT CRASH INJURIES

As I have said, the philosophy of injury prevention has been changing, and instead of stressing adaptation of human beings to machines, we have started building machines that will minimize the consequences of human error. Our work in Los Angeles during the past 10 years has included—though it has by no means been confined to —the performance of controlled, full-scale automobile collisions. We tried making investigations on the highways and streets, with the cooperation of the police, but we weren't successful. Please don't think that I am criticizing anyone. There is a great deal of value to be gained from mass statistics properly analyzed, and the Cornell University group is doing just that. We merely found that a technic of that sort wasn't suited to our facilities and interests, and we retreated to our laboratories to find out what we could discover there.

We are indebted to the U. S. Navy for the site on which we conduct our experiments—an unused airstrip at the Long Beach Naval Station. Our cars are full scale and into them we put anthropomorphic dummies so that we can observe, as accurately as possible, what happens to human passengers in vehicles that are involved in crashes of various sorts. They are patterned after tables that two German scientists made up, late in the last century, after taking measurements of many, many cadavers. We know when, where and, with rare exceptions, how our collisions and other smash-ups will occur, so we can arrange to take motion pictures of them, some of them with "slow-motion" gunsight cameras capable of exposing as many as 200 frames per second. Subsequently, we study and analyze the pictures.

Early in our investigations, we found:

1. Unyielding protuberances on the dash of an automobile are especially hazardous.

2. Unyielding protuberances elsewhere on the car interior are only slightly less hazardous—e.g., in one collision experiment, a rear-seat "passenger" received severe head injuries through violent contact with the ashtray receptacle on the back of the front seat.

3. Through car collisions with unyielding fixed objects, the forces impressed upon all car occupants far exceed the resistance power of any human being.

4. In certain types of collisions, spin forces are more than sufficient to eject the car's occupants violently.

5. Properly designed and installed seat belts have high promise in reducing deaths and serious injuries.

6. Certain other restraining devices, such as a combination lap belt and shoulder harness, provide considerably greater protection than do lap belts alone.

7. The seats in the vehicles used in the experiments were inadequately anchored. That is to say that seat anchorages ruptured at well under the G loadings commonly encountered during 20-25 mph. collisions with fixed objects.

8. The use of pot metal and knifelike shapes for horn rings, and the placement of pointed ornaments on the ends of steering posts unnecessarily expose drivers to severe facial injuries.

In the highway-accident studies conducted by Moore *et al.*, the findings reported in points 1, 2 and 5 have been validated. Other important findings in these studies relate to door locks, rear-view mirrors and steering wheels. Many of these features are receiving or have received serious and skilled attention from the automotive industry. If, to some people's way of thinking, the automotive industry isn't moving fast enough, it should be pointed out that enthusiasm for traffic

safety isn't exactly a characteristic of the driving public. The sparing use of seat belts provides an example, for the best estimates indicate that no more than 2 per cent of U. S. cars have been equipped with them. In Sweden, by way of contrast, the figure was more than 30 per cent several years ago, and the percentage has been rising rapidly since that time.

In his pioneering experiments, Stapp demonstrated that man, properly restrained, can tolerate G forces in the 45-50 range. Most automobile seat belts now in use are designed for approximately 20 G. The question we must decide is whether to try for a greatly accelerated rate of acceptance of the existing 20 G restraints, or to upgrade the design to 30 G, together with attempting to accelerate public acceptance of them. I suggest the latter, but with either we are sure of achieving a substantial reduction in motorist fatalities and injuries.

EACH TRIP TO THE GROCERY IS HAZARDOUS

One of the worst misconceptions of the motoring public is that danger is negligible at the low speeds that commonly prevail to neighborhood trips to stores, clinics, offices and so forth. Apparently the many local trips made without mishap have provided drivers and passengers with the assurance that nothing serious can happen within their own neighborhoods. To the extent that they think about the matter at all, they undoubtedly arrive at this conclusion on the basis of the slowness with which they must drive within city limits. In our investigations, however, we have found that forces in the 25 to 30 G range are regularly produced when automobiles are driven into fixed barriers at speeds of 20 to 25 mph. Very few human beings have the strength to resist a force in excess of 1 G, and not even that much at the rates of application encountered in collisions.

During such events, the motions of car occupants are violent and completely involuntary because of the overwhelming forces that result from rapid decelerations. The initial impact or contact in many intersection-type collisions often leads to a loss of control of one or both cars. A secondary collision is thus guaranteed in such cases, and it may be with another car, a building, a utility pole or—worst of all—a group of pedestrains on a public sidewalk. The driver wearing a seat belt, on the other hand, always stays behind the steering wheel, where he can either retain or regain control quickly. As can readily be proved by documented accident-investigation reports, there is nothing farfetched about the positive arguments for seat belts.

WE SHOULD TEACH DRIVERS HOW TO USE SPEED

The dilemma of street and highway design engineers and of traffic engineers is perhaps the most perplexing of all. A multiplicity of decisions must be made on design speeds; width of lanes, shoulders, right-of-way medians; vertical and horizontal alignments; geometry of access and egress roads, including the lengths of acceleration and deceleration lanes; and signs, signals and road markings. The maximum design speed promulgated by the American Association of State Highway Officials is 70 mph. The maximum legal speed in the vast majority of states is 65 mph. or less. Thus, we can say that there is consistency. But automobile speed capabilities are something else, in that most American and many foreign-built cars have steady-state speed capabilities of 90-100 mph. or more. As has already been implied, substantial numbers of drivers utilize these higher speed capabilities, and consistency has now largely vanished. In spite of this situation, however, it has been shown that high-grade facilities such as freeways, expressways and turnpikes have accident records that are very substantially and consistently better than those of lower-grade roads. If part of the effort on speed reduction or limitation were devoted to educating and training our population on how properly to utilize high speeds, we could look forward to long-range improvement in highway safety. To the extent that it is currently feasible, this is being done in approximately half of all our secondary schools.

Among the many other tasks that they perform, highway engineers are more or less continuously improving the alignments of older roads. It is common knowledge that many, if not all, of the original alignments of those older roads followed animal trails and river courses. In changing the alignments, the objectives of the engineers are higher and more uniform speeds of travel, more economy and greater safety. In some worthwhile measure, they always reach the first two objectives, but not always the third.

Another strong trend is toward wider median strips on divided highways, and toward construction, in the medians, of barriers to prevent crossovers by cars that have gone out of control. The requirement that a barrier must meet is not merely that of strength, although this is one of the desirable characteristics. Additionally, it must deflect out-of-control cars back into the traffic stream, and so far as possible bring careening cars to a stop without serious injury to the occupants. The last of these requirements cannot be met by the highway designer alone. Car designers and the motoring public must do their share. The reluctance of the public to use restraints is the least promising aspect of this cooperative endeavor. We can, perhaps, learn a lesson from the regimen pursued in Sweden. There, the medical profession has played, and continues to play, a major role in convincing the motoring public of the merits of driver and passenger restraints.

"They Don't Build 'Em Like They Used To"

KARL M. RICHARDS
DETROIT, MICHIGAN

BEFORE I START giving you my prepared remarks, I'd like to say that I have made two appearances before the Roberts Committee of the House of Representatives recently in opposition to the proposal for federally-imposed vehicle-safety requirements that Dr. Horace Campbell has made here. It would take too much time for me to answer him completely, but let me point out one fact. The Great White Father has never done much for industry, and specifically hasn't done a great deal for safety—not as much as some people think. The automobile industry has developed a procedure for establishing standards that provides a flexibility that it wouldn't possess under a federal standard-setting agency.

Now, Mr. Roberts was very much surprised when he first brought up his bills and found that several federal departments opposed them. Their reason was that the automobile makers have been cooperating with their people over the years, and they realize that if they were required to replace the Society of Automotive Engineers, the American Standards Association and other technical groups that have been setting specifications, they would have to duplicate already existing research facilities costing well over half a billion dollars, and to avoid setting barriers in the way of industry, they would have to call on the help of the very men who are now doing the same work for the automobile manufacturers.

As I told Chairman Roberts, before testifying before his committee I had a meeting with the European manufacturers, and when I mentioned my upcoming trip to Washington, they were appalled. They told me that one of their reasons for visiting Detroit was to hear about our standards-setting procedures in the hope that they might find a way of escaping the dictates of their respective bureaucracies, the staffs of which are well-meaning professors without practical backgrounds.

AMERICA'S TRAFFIC-SAFETY RECORD IS COMPARATIVELY GOOD

We are here today to discuss traffic safety under the singularly appropriate title "The Crash," and although we can think of traffic safety in such simple terms, it is an amazingly complex problem. Every day, on our streets and highways, some 70,000,000 vehicles are maneuvered through the intricate maze of modern traffic. The vehicles are controlled by independently thinking individuals with varying degrees of ability and training who must meet and cope with dozens of unique driving situations in every mile that they drive.

Fortunately, these drivers, like a well-known brand of soap, have a nearly perfect percentage of purity, and only a very minute proportion of those billions of traffic situations result in accidents. But unfortunately, that small proportion accounts for some 28,000 crashes every day and must therefore be a matter of grave concern.

Scientists and safety specialists have come to realize how little we actually know about all of the variables involved in these crashes. Increased research is desperately needed, but I think you must agree that a tremendous job has been done with the limited knowledge already available. For instance, during the past 25 years the traffic fatality rate per 100,000,000 miles driven has been reduced by two-thirds, dropping from 15.9 in 1935 to 5.3 in 1960. At the same time, the number of registered vehicles has tripled.

This is a record that we can be proud of, particularly when viewed in the light of experience in the other countries of the world. Reports show that although we have two-thirds of the world's vehicles and drive three-fourths of the world's mileage, we have only one-third of the world's traffic fatalities. These figures constitute a highway safety record that is six times better than that of the rest of the world.

OUR PROBLEM DRIVER IS THE AVERAGE ONE

With this good record, improvement in the future will become increasingly difficult to attain. Perhaps the greatest enemy to traffic-safety improvement today is the fact that most drivers realize that they have good records and therefore assume that they are not a part of the problem. Statistically, the average driver can expect to drive for more than five years without a property-damage accident, and for about 40 years without an accident involving a personal injury. He could expect to drive through 40 lifetimes—2,000 years—without an accident involving a fatality.

As a matter of fact, 97 per cent of our drivers have 85 per cent of our accidents. Thus the big job ahead will be to convince the *average* driver that he is a part of the problem. It isn't the other fellow who gets into all of the accidents. A government study has shown that if all accident repeaters were taken off the road, the total of our

Mr. Richards is manager of the Field Services Department of the Automobile Manufacturers Association.

accidents would be reduced by no more than 4 per cent.

Experience over the past 25 years has demonstrated that although there is no panacea for our traffic safety problems, well conceived and intelligent plans of attack can do much to alleviate them. In my remarks I shall try to summarize the important discoveries of the past few years regarding the nature of our traffic-accident problem, and to point out some of the steps being taken by the motor vehicle manufacturers not only to prevent accidents but also to help keep people from being injured in them.

It is difficult to separate the motor vehicle from the other two elements in the traffic safety equation—the highway and the driver. Yet, because of the push of competition and the lift of voluntary cooperative programs, the vehicle has been improved much faster than has either the highway or the driver. It is popular in some circles to place the major blame for highway accidents on vehicle design, and to demand that the manufacturers solve the problem. Such an approach is not only ill advised but could be dangerous to the cause of safety.

POSTWAR IMPROVEMENTS HAVE MADE CARS SAFER

In "The Federal Role in Highway Safety," a report to Congress from the U. S. Bureau of Public Roads, the conclusion is reached that "Today's motor vehicles are structurally safer than any that have heretofore been manufactured, and the industry is giving attention to many features of the vehicle that add to its safety." This statement can be supported factually. Both National Safety Council data and studies by the Cornell Automotive Crash Injury Project document the fact that today's automobiles reduce by one-third the risk of serious or fatal injuries to car occupants when a traffic accident takes place.

A major safety advance of recent years has been the modern automobile design itself, with its lower center of gravity, larger glass area and wrap-around windshield for better vision, and better handling and riding qualities. Also worth special mention are three major postwar advances in vehicle headlighting and the various new power assists for drivers.

The increased horsepower of modern cars came in for some criticism a few years ago. However, the industry was convinced that prewar cars lacked adequate reserve power for safe operation on today's congested highways, and that the added horsepower or maneuverability would be a positive safety factor. The facts show that this industry view was correct. The Bureau of Public Roads study showed, on the basis of highway surveys in many different parts of the nation, that high-horsepower and high-performance cars have distinctly better safety records than do low-powered and low-performance cars.

These have been a few of the areas in which motor vehicle makers have accomplished significant gains to aid the driver in avoiding accidents. Perhaps even more pertinent to this discussion is the work that the vehicle producers have done in reducing the likelihood of serious injury to a motorist or his passengers if an accident should occur.

Much of this work has been based on information obtained in hundreds of scientifically-staged accidents at automotive proving grounds. These carefully planned accidents continue to turn up new facts about the behavior of both vehicles and their occupants during crash impacts. Using full-scale vehicles, auto safety engineers have duplicated nearly every type of highway crash situation. Remotely controlled cars traveling at highway speeds have been crashed into wooden, concrete and metal barriers. Others have been rolled over, rammed head-on or broadside by other vehicles, and even struck by a giant pendulum. Every crash has been recorded from start to finish by means of elaborate electronic instruments and by ultra-highspeed motion picture cameras for later analysis in slow-motion. Many of the test vehicles have been "manned" by anthropomorphic dummies approximating human beings in size, weight and movement. Carefully instrumented, they have aided in determining the exact sequences of collision events, and have disclosed the injury-producing contacts with objects on the interiors of the vehicles.

Although the crash-test findings are providing first-hand information on vehicle collisions, they are also proving that motor vehicles do a lot to reduce accident dangers. Engineers, once criticized for structural damage to vehicles in accidents, are able to show that this same bending and yielding safeguards passengers by absorbing much of the abrupt shock of a crash.

For impact speeds above 15 mph., the deceleration level in the passenger compartment varies from one-half to one-third of that experienced by the front of the vehicle. This fact indicates that the front structures of present cars are substantially effective energy absorbers. Naturally, work in this important area is being continued.

Simple crash and roll-over tests were conducted by industry engineers as early as 1933. The greatest share of the work in this field, however, has been accomplished since 1953, following the development of new electronic instruments for measuring and recording crash information. In addition to measuring the over-all effectiveness of vehicle safety features, the tests have also provided a special proving ground for new safety devices. Some of the first tests measured the effectiveness of various types of safety door latches under actual crash conditions, and led to their adoption as standard equipment by the industry in 1956. Crash studies have also aided in the de-

velopment of recessed-hub steering wheels, automotive seat belts, better seats and seat-cushion retention, improved instrument panel designs, stronger roof-support pillars and more effective energy-absorbing safety padding.

Data provided by full-scale tests have enabled vehicle engineers to build laboratory equipment that can simulate conditions encountered in actual crashes. These special research facilities are used extensively for individual studies of new safety features. Related crash-testing activities in the industry have demonstrated that removal of roadside hazards such as trees and deep ditches and the adoption of new approaches to the design of protective barriers and utility and sign poles can greatly reduce highway accidents.

Much of the information found in the crash tests has been widely distributed through the automotive industry by reports to professional societies, government agencies, safety organizations and the Vehicle Safety Committee of the Automobile Manufacturers Association.

AUTO MAKERS HELP FINANCE INDEPENDENT INVESTIGATIONS

Most of the crash testing is conducted at automotive proving grounds, but the auto makers also sponsor or cooperate in similar tests by outside organizations. Many of these studies are related to the tolerance of the human body under various impact conditions. The industry maintains a double check on its vehicle-safety progress through supporting the crash-injury research program of Cornell University. Annual grants through the Automobile Manufacturers Association to that program help make possible the collection and analysis of accident records from 22 states.

Augmented by photographs, the reports give detailed descriptions of both vehicle damage and passenger injuries. In addition to studies made by the researchers at Cornell, vehicle manufacturers obtain duplicate copies of the reports involving their respective makes of vehicle for further analysis and for the evaluation of model changes and improvements.

We admit freely that we have a very selfish interest in the safety of motor vehicle operation on the nation's streets and roads. For us, traffic safety is good business, quite aside from its humanitarian values. And because traffic safety involves three elements—the vehicle, the highway and the driver—our industry has never limited its concern for safety to the vehicle alone. We have endeavored constantly to give effective support to every phase of the broad and complex work of traffic-accident prevention.

Numerous long-standing relationships exist between the industry and the other organizations active in traffic-safety fields, including the Committee on Engineering and Vehicle Inspection of the American Association of Motor Vehicle Administrators, the Safety Section of the Bureau of Motor Carriers of the Federal Interstate Commerce Commission, and the National Committee on Uniform Traffic Laws and Ordinances.

Currently, the industry makes annual grants of nearly $2,000,000 to national organizations and universities working on traffic safety programs of various types. These grants, largely channeled through the Automotive Safety Foundation, in Washington, cover such aspects as training of traffic engineers and traffic police, driver training and licensing programs, support of the National Safety Council, and many research projects on the human factors in accidents.

With respect to the vehicle, the development and testing of vehicle-safety engineering and devices involves technics and procedures that have evolved over the years and have been proved through vast experience. Cooperative work with officials charged with the public responsibility for traffic safety is an essential part of this procedure.

CONCLUSION

Any arbitrary interference with the established, time-proved and highly successful procedures that use both competitive forces and cooperative programs to assure maximum progress in vehicle-safety engineering would be highly undesirable. Safety engineering by law would tend to freeze design and to shut off future research and improvement.

The way must be left open for continued development of new ideas, new devices and new technics for greater safety. Each of these will be carefully studied and tested, and those that prove effective submitted to the driving public for its increased protection. This approach, balanced by improvements in other areas of the traffic-safety picture, offers our greatest hope for continued accident reduction.

METHOD OF BLOCKING DRUG-RESISTANCE

A new method of fighting bacteria which resist antibiotics was described in the November 18 J.A.M.A. by John K. Lattimer *et al.*, of New York City. They told of noting that drug-resistant bacteria produced a high level of urease, which, like all other enzymes, acts as a catalyst in the chemical processes of the body. Experimentation revealed that chlormerodrin, a mercurial compound, interfered with the production of urease and rendered the bacteria susceptible to the same antibiotic that they had previously resisted.

Subsequently, two other drugs were developed that blocked the enzyme's activity more effectively. These are still in the experimental stage and haven't been named. The enzyme blockers were most noteworthily effective in rendering Aerobacter aerogenes susceptible to antibiotics. That microorganism is responsible for a large share of chronic urinary infections, the authors said.

Questions and Answers

Following the Panel Discussion on Auto Crash Injuries

Unidentified Questioner: Some entirely contradictory evaluations of the safety aspects of the new cars have left us confused. Dr. Horace Campbell has said one thing, and Mr. Richards has said another. Perhaps neither has convinced the other here today, and we'd like each to be given a chance for rebuttal.

Dr. Horace Campbell: The best thing I can do, I think, is to refer you to the sheets that were distributed to you (Figure 1). The devices that are described there were suggested several years ago by a responsible segment of the motor car industry, and I point out that none of them is to be found on any of the cars that crowd our highways today. Any one of those proposals is utterly sound, but they are just on paper.

In regard to my call for federal legislation, let me say this. If all of the items on that sheet of paper put out by Ford in August, 1958, had been built into the subsequent new car models, I wouldn't be asking for federal standard-setting today. The point is that these are the things that the medical profession has asked of the automobile industry for the past 25 years. What else can we do? We've talked and we've begged. Dozens of doctors—not just I—have written papers year after year, and what do we get? We get cars that literally are no safer than those that were manufactured 25 years ago.

The statements that Mr. Richards has cited in an attempt to convince us that the car is so safe today, I must say frankly, are just not true. The proof is the way that it performs in a crash. Now I grant that certain features are safer. The doors don't open quite so readily as they used to—but only 35 per cent less readily. The designers ought to have achieved a 50 per cent improvement, or perhaps 90 per cent.

Perhaps the most convincing evidence of the inadequacy of today's automobile may be obtained by comparing it with Liberty Mutual's SURVIVAL CAR II. Even this car, I think the designer, Mr. Frank Crandell, would agree, is something like half the car that we will eventually get. As for Mr. Richards' contention that the Railway Safety Act has hampered or impeded the creation and installation of safety technics by the railroads, his assertions don't agree with what I have learned from railway men during the last 50 years. There were 12 passenger deaths in 1959.

Mr. Richards: I don't think we need apologize for the improvements that have been made in the vehicle over the years. The chances of being injured or killed in a vehicle, as I pointed out, have been reduced by one-third since 1941. Now that represents a tremendous improvement. We don't feel that we are building the ultimate vehicle today. We're a long way from that. If I may turn the tables for a moment, I might remind you that the work that has been done in finding cures for cancer and for the common cold hasn't been completely successful.

For years, people have been telling us what we should do, but there's a vast difference between just an idea and something that can be incorporated into a vehicle. Within the industry, we have safety engineers who have never had an idea of theirs put to use. They are retained because they stimulate ideas. On the other hand, even the most practical of safety features are no good to us unless the public will buy the cars that are so equipped. The current infrequency of accidents—a property-damage accident every five years, an injury-producing accident every 40 years, and a death-producing accident once in each 2,000 years—is the reason why people aren't willing, for example, to ride backwards in automobiles, even though they would reduce their chances of serious injury by doing so.

I think a tremendous job has been done, and a fine job will be done in the future—probably more rapidly because we have more testing and research facilities today.

BRAKES AND STEERING RATIOS—ADEQUATE OR INADEQUATE?

Dr. John F. Kelly, Fort Dodge: How about some things to forestall or prevent accidents? I am critical of several features in the present-day automobile. First, I think that in the past five years we've seen a decline in the quality and effectiveness of brakes in American cars. In 1956, for example, the Chrysler Windsor had 251 sq. in. of brake lining and a 225 hp. engine; in 1960, it had 218 sq. in. of brake lining and a 305 hp. engine. The weight of the car hadn't changed substantially. Second, there is the matter of steering ratios. The ability to maneuver an automobile is a very critical factor, and the steering ratios in present cars are quite high, requiring many turns of the steering wheel to produce a proportional change in the course of the car.

It seems that manufacturers concentrate on more superficial qualities in an automobile.

Mr. Mathewson: Accident prevention is something of a will-o-the-wisp. The only person, really,

who can prevent an automobile accident is the driver. Naturally enough, the highway designer contributes, in either one way or another, and the same can be said of the automobile designer. I think, after all that has been said, that our present cars are relatively safe as compared with the older models.

If a vehicle weighing X pounds is moving along a roadway at a certain velocity, there is a limit to the promptness with which it can be brought to a stop. We have thought of installing a device to produce counter rocket thrust as a means of stopping runaway trucks—over-the-road rigs that characteristically run away on some of our California grades, and on those of some other states as well. The thing can be done. As a matter of fact, we could put on counter rocket thrust that no human being could stand up to.

Let's recognize that "John Q" wants to sit in his automobile with a great deal of freedom. As of now, he refuses to wear a seat belt that would restrain him and would decrease his speed at approximately the rate of the portion of the car in which he is sitting, in case of an accident. Thus, he could take advantage of the front-end collapse distance to which Mr. Richards has referred. Our experiments, incidentally, have shown that this front-end collapse is very, very good in some cars.

Let's take, by way of contrast, what has been accomplished in Sweden. From a source there, I've learned that the Swedes are approaching a point where 50 per cent of their people possess seat belts in their cars and wear them regularly. In the United States today, the figure is something less than 2 per cent, and perhaps less than 1.

We are going to see changes come about. Sure, everyone needs prodding once in a while. The industry has had this prodding, and it is responding to it. The same is true of the highway engineer. There isn't any shortcut to a solution of the problem.

Mr. Richards: I'd like 30 seconds for a reply to Dr. Kelly's remark about brakes. Yes, there's a little misunderstanding in this area. The State of Pennsylvania mistakenly set up a law establishing the minimum number of square inches for brake surfaces. I brought the officials from that state to Detroit and showed them that the whole trend is toward less brake surface but more effective braking. We don't like to see design features frozen into law, for then industry research is limited.

Proving-ground test data proved conclusively that there has been a gradual and very important improvement in braking over the years—something that I don't think has been true of every other feature of the vehicle. Every year we build a safer vehicle than the year before.

Dr. Kelly: As optional equipment, the manufacturer offers larger brakes with more brake lining. I interpret that as meaning that the manufacturer considers the larger brakes preferable. That's all I have to go on.

Dr. Werner Pelz, Charles City: I think that neither Mr. Mathewson nor Mr. Richards gave us the answer. Here, Dr. Kelly, is a specific reply to your question. At sports car races in Florida two years ago, a man connected with Chevrolet had the fastest car there. It was also the car with the biggest engine. But within two rounds of driving on the road course, that car had to quit. There was just one reason: it had run out of brakes. Our brakes aren't getting better; they are getting worse!

You mentioned power equipment. The advertising agencies would have us believe that it is an improvement, but it is not. The wheels are getting smaller, and consequently the brake drums and brake linings are smaller, providing less effective braking area. If you don't have enough brake lining and haven't ample space for cooling, you will have fading, and if this happens, you'll have no brakes.

Another thing. Take any two automobiles and bring them to a stop from, let's say, 30 mph. One car will stop in half the distance that the other will.

Ford proved, in 1958, that safety features are difficult to sell because they are expensive. Money is a part of this problem.

I wish the panel would elaborate a bit about the rationale of the new compacts in which one has to turn the wheel six turns in order to achieve the maximum change of course.

Mr. Russell Brown, Washington, D. C.:* The answer may be for the car manufacturers to make safety features standard rather than optional equipment, but it would be necessary for all of the makers to begin using these features simultaneously. The Chrysler management has told me that it could put on a muffler that would last the life of the car, but aluminizing mufflers costs money and overall prices must be kept competitive.

Dr. B. J. Campbell: Even if seat belts were standard equipment—as was the case in some 1958 cars—only about one-third of those who had them would use them. Thus, I suppose that it would by no means be a complete solution just to make sure that seat belts were present in all cars. But if one-third of those who had them used them—i.e., if the rate of utilization remained constant—one-third of a big number would be preferable to one-third of a small one.

I think that people must be educated to want these things and not to resent them. I've heard people commenting unfavorably about the padding in their cars and asking where they could get ones without padding. Thus, it seems to me that especially when the use of a device depends

* President of the Insurance Institute for Highway Safety, and moderator of this discussion.

upon cooperation by the individual, we have an educational job to do.

SHOULD MAKES OF CARS BE GIVEN SAFETY RATINGS?

Dr. C. W. Maplethorpe, Toledo, Iowa: What would be the advantages and disadvantages of rating cars according to safety? Statistics might be compiled on fatal car accidents by make of car, and so that the question of good or poor maintenance might be avoided, figures could be kept only upon the current models of each make. If cars of a particular make made up 25 per cent of the new cars that had been sold but had been involved in 30 per cent of the fatal accidents involving new cars, the public might be so informed.

Mr. Brown: I'm not quite sure. Perhaps people who buy a particular make of car are less accident-prone than are those who buy other makes. Thus it seems that credit or blame would be assigned unfairly under such a rating system.

Mr. Richards: I don't think that ratings on that basis would serve to evaluate the safety features on the automobile at all. This proposal is based on the fallacy of blaming the vehicle for the accident.

Dr. Horace Campbell: I'm sure you know that I think it's a peach of an idea, if the car companies really have the guts to allow it.

Dr. B. J. Campbell: Some consumer publications, I understand, have attempted this sort of thing. But I agree with Mr. Brown. I don't know what kind of person buys a particular make of car, but personality seems to enter into every other sort of choice and it could be a factor here too. The question could be studied, though.

Unidentified Questioner: Should one put seat belts into a convertible?

Dr. B. J. Campbell: The chances of a roll-over accident are about one out of five, and I suppose that you are concerned about that possibility. In the other four out of five possibilities, the seat belt presumably would be as beneficial in a convertible as in any other type of car. If one were to roll a soft-top convertible, it would be bad if he were thrown out and it would be bad if he stayed in. But with the new low-center-of-gravity, the chances of rolling have been reduced, and I'd have the belts installed.

Unidentified Questioner: Is there a significant difference in safety between rear-engine and front-engine cars?

Mr. Richards: Well, I don't think we have any statistics on that subject. There's a certain amount of protection that comes from the collapse of the front end of the vehicle. I think that in the tests that have been run, the results have probably been equal. Also, the rear-end engines seem to give good protection to passengers in accidents involving the back of the car.

SUMMARY STATEMENTS

Dr. Horace Campbell: I'm going to ask Iowa doctors to stand up and scream for safety devices. I'm going to ask them to go to their car dealers and point out that the models on their showroom floors lack crash padding. Then I want them to tell those car dealers, as pointedly as they can, that they aren't going to buy new cars this year for that reason. Believe me, if enough people do that, it will do a lot of good!

Mr. Mathewson: The doctor-patient relationship is unique, and it is valuable. I feel that the principal reason why Sweden has had such a large degree of success in getting people to install and use seat belts is that the doctors there have advised their patients to use them. Thus, you Iowa doctors, when you talk to a patient and to the relatives of a patient who has been injured in an automobile accident, make the most of your opportunity to tell these people how they can protect themselves.

OBSTETRICS AND GYNECOLOGY COURSE AT IOWA CITY

Tuesday, January 9, 1962

2:00 "Ectopic Pregnancy, a Constant Diagnostic Problem," Robert H. Barter, M.D., Professor of Obstetrics and Gynecology, George Washington University, Washington, D. C.

2:30 "Experience With Radioactive Gold in the Treatment of Ovarian Malignancy," W. C. Keettel, M.D.

3:00 "Premenstrual Tension," Frederick J. Stoddard, M.D., Milwaukee, Wisconsin

4:10 E. D. PLASS MEMORIAL LECTURE: "Telemetering of Ovarian Function, Experimental Approach to the Electronic Detection of Ovulation," S. Leon Israel, M.D., chairman, Dept. of Obstetrics and Gynecology, University of Pennsylvania, Philadelphia

6:00 SOCIAL HOUR AND DINNER—University Athletic Club

Wednesday, January 10, 1962

9:00 "Review of Departmental Activities," Dr. Keettel

9:30 "Hormonal Changes in Normal and Abnormal Pregnancy," James T. Bradbury, Sc.D.

10:00 "The Psychophysical Indications for Hymenal Dilatation," Dr. Barter

10:45 "Hirsutism as a Gynecologic Problem," Dr. Israel

11:15 SYMPOSIUM—Ovarian Conservation
Drs. Barter, Israel, and Stoddard

12:30 LUNCHEON—Doctors' Dining Room

1:30 "Detection of a Pressor Substance in Toxemic Patients," William F. Howard, M.D.

2:00 "Chronic Trichomonas Infection," Dr. Stoddard

2:30 SYMPOSIUM—Management of Third Stage of Labor
Drs. Barter, Israel, and Stoddard

3:45 "Pre-term Delivery of Rh Sensitized Patients," C. P. Goplerud, M.D.

Antepartum Venous Thrombosis With Multiple Pulmonary Emboli: A Case Report and a Discussion

CLIFFORD P. GOPELRUD, M.D., AND GEORGE S. ANDERSON, M.D.
IOWA CITY

THROMBOPHLEBITIS of the deep veins of the legs is a rare antepartum complication. Ullery[1] reported an incidence of one in 5,000 deliveries at the Philadelphia Lying-In Hospital, and listed other incidence figures as high as one in 1,000 deliveries.

Mrs. D. O., a 28-year-old gravida VI, para II, aborta III, was seen on December 29, 1959. One of us (G. S. A.) had previously treated her for proved myxedema. Roentgenograms of the chest at the time of her thyroid studies one year previously had been read as normal. Electrocardiographic findings at that time had been consistent with myxedema. The first pregnancy that went to term had been uneventful, and except for a moderately long labor, there had been no complications of labor, delivery or puerperium. During the second successful pregnancy, a mild hypertension had occurred near term, and there had been a superficial thrombophlebitis of the left leg during the puerperium.

The complaint on admission was of a dull substernal pain that radiated to both shoulders and was aggravated by deep breathing. It had been present for five days, along with shortness of breath and a slight non-productive cough. Her last menstrual period had begun on November 29, 1959.

On admission, the patient was hyperventilating. Her skin was warm and pale, her blood pressure was 140/80 mm. Hg, her pulse was 100 beats per minute, and her temperature was 99.2°F., orally. Her chest was clear to percussion and auscultation. There was a widely split mitral first sound, but no other abnormal heart sounds. The abdominal examination revealed no abnormalities. There was squeeze tenderness of the left calf, but Homan's sign was negative and remained so. No evidence of superficial thrombophlebitis was found. Roentgenograms of the chest were normal. The R wave in AVR was very prominent, but no other electrocardiographic evidence of abnormality was seen.

The results of the laboratory work performed on admission were as follows: VDRL negative; urinalysis negative; hemoglobin 9.4 Gm. per cent; hematocrit 35 per cent; white blood count 9,700/cu. mm., with 76 per cent segmented polymorphonuclear leukocytes; and serum glutamic oxalic transaminase 37 units/ml.

During the next five days, the patient continued to complain of shortness of breath and chest pain. The pain was not relieved by narcotics, sedatives or combinations thereof. On January 2, 1960, the protein-bound iodine was 4.3 micrograms per cent, and the reticulocyte count was 2.2 per cent.

After one episode of pain on January 3, the blood pressure dropped to 80/60 mm. Hg, and the patient developed a systolic gallop. The subsequent morning, her temperature rose to 102°F., orally. On examination of the lung fields, there were crackling rales noted in the right lower chest. Within 12 hours, a pleural friction rub was heard in this area.

On January 4, the hemoglobin was 9.2 Gm. per cent, the hematocrit was 38 per cent, and the serum amylase was 69 Somogyi units. During the ensuing two days, rales and friction rubs were heard in various areas of both lung fields. On January 4, fluoroscopy of the chest revealed a patchy increase in the density of the base of the lateral aspect of the lower lobe of the left lung. Treatment with heparin intravenously and intramuscularly was initiated on that date. An electrocardiographic evaluation on January 5 revealed minor T-wave changes in AVF, but there was no evidence of acute elevation of the right ventricular pressure. On January 5, the serum glutamic oxalic transaminase was 60 units/ml., and on January 6, the serum iron was 50 gamma per cent, the iron-binding capacity 140 gamma per cent, and the iron content was 23.6 per cent of saturation.

On January 8, portable roentgenograms of the chest revealed a further increase in the extent of the density in the base of the left lung, having the appearance of fluid in the pleural cavity. There was a fairly well defined area of increased density in the lateral angle of the right middle lobe, and another extending laterally and slightly upward from the right hilus.

The patient improved slowly until January 13, when she developed sudden pain in her left eye along with marked lacrimation. Within an hour,

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proptosis with orbital edema and ptosis of the left eyelid was noted. Conjunctival edema and limitation of rotation were present. She was examined by the ophthalmologic consultant and it was his impression that there was venous thrombosis—probably cavernous sinus thrombosis. Within four hours following the onset of pain in the left eye, similar signs and symptoms were noted in the right eye. But 24 hours later, all the symptoms and signs referable to the eyes had disappeared.

Oral anticoagulant therapy with warfarin sodium was started on January 17, and within 24 hours the prothrombin time was in therapeutic range. Heparin therapy was discontinued on January 18.

On January 25, P-A and lateral roentgenograms of the chest showed two new areas of infiltration in the first and second right anterior rib interspaces that had not been apparent on the film taken January 8. There was an irregular infiltrate present in the third anterior rib interspace that was then more readily apparent than on January 8.

Because no menstruation had occurred subsequent to the period beginning November 29, 1959, it was thought that the patient might be pregnant. A biologic test for pregnancy was positive on January 26, 1960.

On February 1, the patient was discharged from the hospital to continue therapy with warfarin sodium, and she was instructed to wear elastic bandages on both lower extremities. She was examined intermittently during the ensuing month and showed gradual improvement. The prothrombin time was checked at intervals and remained in therapeutic range.

She was examined by the second observer (C. P. G.) for the first time on March 2, 1960. Then, physical examination revealed an obese but fairly healthy-appearing female 65 inches tall, weighing 195 pounds. Her heart was not enlarged, the rhythm was regular, and there were no murmurs. The blood pressure was 134/66 mm. Hg. The lungs were clear to percussion and auscultation. The uterine fundus was measured 17 cm. above the symphysis pubis. There were mild varices in both lower extremities, and there was mild tenderness in the right calf. The uterus was symmetrically enlarged and softened, in accordance with the duration of amenorrhea. The adnexal regions were normal. The cervix was well epithelialized, and cervical cytology revealed no abnormal cells. The pelvic measurements were within normal range. A urinalysis showed no abnormality. The hemoglobin was 8.8 Gm., and the hematocrit was 31.5 per cent.

The uterus enlarged at the expected rate. The heart tones remained good throughout the pregnancy. There was a period of time in June and July, 1960, when she had moderate edema, but it responded well to treatment with an oral diuretic. Her greatest weight was 217 pounds, reached about three weeks prior to delivery. Her blood pressure ranged from 70/50 to 134/66 mm. Hg.

Warfarin sodium therapy was stopped on April 18. The hemoglobin on that date was 7.8 Gm. per cent. Because of the laboratory information obtained during her hospitalization, it was felt that this represented an iron deficiency anemia. Oral iron therapy was initiated on April 18. On May 11, the hemoglobin was 9.9 Gm. per cent, the hematocrit was 35 per cent, and the reticulocyte count was 3.1 per cent. On June 15, the hemoglobin was 12.5 Gm. per cent, and the hematocrit was 39 per cent.

The patient was admitted to the hospital in labor on September 7, 1960, and had a spontaneous delivery of a 3,715 Gm., normal-appearing male whose Apgar rating was 8. The total labor was 6 hrs. 26 min. She was ambulatory within 12 hrs., and wore elastic bandages on both lower extremities during her stay in the hospital. The postpartum course was essentially uneventful, except for superficial thrombophlebitis which developed in the left calf on the second postpartum day. Her temperature was not elevated during her hospitalization. On September 10, her hemoglobin was 14.6 Gm., and her hematocrit was 46 per cent. She was discharged in good condition on the seventh day after delivery, and was instructed to wear elastic bandages on both lower extremities.

When the patient was seen for the six-weeks postpartum examination, menstruation had not been established, and the physical and pelvic findings were normal.

DISCUSSION

The incidence of pulmonary embolization in 200 reported cases of antepartum deep thrombophlebitis is 18 per cent. With this high incidence of embolus, it would seem that anticoagulant therapy is indicated, provided that there are no serious maternal or fetal complications. Quick[2] has fed dogs therapeutic doses of bishydroxycoumarin

TABLE I

INTRAUTERINE DEATHS DURING PROTHROMBINOPENIC DRUG THERAPY

Author	Number of Deaths Reported	Drug Employed
Sachs and Labate	1	Bishydroxycoumarin
Quenneville et al.	3	(1) Bishydroxycoumarin (2 & 3) Bishydroxycoumarin and ethyl biscoumacetate
Blum	1	Bishydroxycoumarin
Epstein	3	Warfarin sodium

during the last week of pregnancy, and their pups have shown hemorrhagic tendencies severe enough so that none survived without vitamin K. On the basis of experiments with rabbits, Kraus *et al.*[3] believe that prothrombinopenic agents are contraindicated during pregnancy. Sachs and Labate[4] have reported intrauterine death in a patient treated with bishydroxycoumarin for 53 days of the third trimester. At autopsy of the stillborn, there was evidence of pulmonary hemorrhage. Quenneville[5] reports three intrauterine deaths that occurred during prothrombinopenic therapy in 33 patients. In two of them, no hemorrhagic lesions could be demonstrated at autopsy, but the intrauterine deaths occurred at a time when the prothrombin time exceeded the "therapeutic range." In the third instance, there was evidence of a recent intervillous hematoma that may or may not have been related to the drugs employed. Blum[6] reported intrauterine death in one patient receiving bishydroxycoumarin that occurred during a short period when the prothrombin time exceeded the "therapeutic range." Epstein[7] has reported three instances of intrauterine death that occurred during treatment with warfarin sodium. At autopsy, however, none of the macerated fetuses showed evidence of hemorrhage.

Feldman and Smith[8] reported the continuous use of bishydroxycoumarin for 32 weeks in a patient who had developed thrombophlebitis during the first trimester of pregnancy. She was delivered without excessive blood loss, and her infant was treated with vitamin K_1 immediately after delivery and had no hemorrhagic difficulty. There are other reports of long-term prothrombinopenic therapy without damage to the fetus.

The heparin therapy of the patient reported here was initiated on the thirty-seventh day after the onset of her last menstrual period, although the pregnancy had not been diagnosed at that time. Prothrombinopenic therapy was initiated on the fiftieth day, and the prothrombin time was adequately prolonged within 24 hours. Such therapy was continued until the 142nd day of the pregnancy. After the embolic phenomena had cleared and there had been no evidence of activity of the disease at the original site for several weeks, the treatment was discontinued.

Any patient receiving anticoagulant therapy may have either spontaneous or traumatic hemorrhagic complications. In obstetrics, the immediate puerperium is the time when such a complication is most likely to occur. In patients receiving prothrombinopenic medication, vitamin K_1 oxide should be given intravenously at the onset of labor, and fresh whole blood should be available in case postpartum hemorrhage ensues. It might be wiser to discontinue prothrombinopenic medication several days prior to the anticipated labor, and treat the patient with heparin, the action of which can be counteracted rapidly and effectively with protamine sulfate.

SUMMARY AND CONCLUSIONS

1. A patient with antepartum thrombophlebitis of the lower extremity, subsequent multiple pulmonary emboli and infarction, as well as possible cavernous sinus thrombosis, has been reported.

2. Antepartum peripheral thrombotic disease is not common, but is complicated by pulmonary embolization in one out of five instances.

3. Anticoagulant therapy is probably indicated in those patients who demonstrate embolic phenomena, in order to reduce maternal mortality, but there are certain maternal hemorrhagic complications that may occur.

4. To date, at least eight intrauterine deaths during prothrombinopenic therapy have been reported. To our knowledge, none has been reported during heparin therapy.

5. The onset of action of heparin is rapid, is of relatively short duration, and can be combatted rapidly if bleeding occurs. It may be the most appropriate therapy near term and in the puerperium. It is probable that it does not cross the placenta, possibly because of its high molecular weight.

6. Prothrombinopenic agents act more slowly, and the time required for nullifying their action is also increased. They are more suitable for outpatient therapy than heparin, but at all times during therapy the prothrombin time should be observed very carefully to make sure that it does not become unnecessarily prolonged.

7. Because of the possible danger to the fetus, prothrombinopenic medication should be discontinued after the embolic phenomena have cleared and the local thrombotic disease has been adequately controlled.

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State University of Iowa College of Medicine

Clinical Pathologic Conference

SUMMARY OF CLINICAL FINDINGS

A 32-YEAR-OLD MECHANIC was admitted to the University Hospitals with a chief complaint of swelling of the face and abdomen and diminished urinary output for 10 months. His first urinary symptom, enuresis, had persisted until he reached 18 years of age. Ten years before admission, the patient had noted right flank plain. Intravenous pyelograms had shown signs of right hydronephrosis, and recurring discomfort in that region during the next eight years led to a right nephrectomy. Pathologic examination of the kidney showed chronic pyelonephritis. Thereafter, the patient had been well for almost two years before he noted edema of the face, abdomen and extremities. In the nine months prior to his entry into the hospital, he had been given adrenal steroids and had experienced intermittent relief of the edema. After several months of this therapy, he had noted epigastric distress which was shown by x-ray examination to have been due to a peptic ulcer. The symptoms subsided after a month of treatment with a bland diet and antispasmodics. One month before admission, a cellulitis of the right arm had made it necessary to stop the steroid therapy. More edema had followed, and the patient was sent to the University Hospitals for care.

Physical examination at the time of admission showed an alert, cooperative and edematous, chronically ill young man. The blood pressure was 150/115 mm. Hg, the body temperature was 98°F. and the weight was 182 lbs. The pulse rate was 76 per minute and the respirations were 20 per minute. The fundi were normal. The chest was dull to percussion below the seventh ribs. Breath sounds were absent in the same area. Crepitant rales at the end of inspiration were present above the area of dullness. The heart was normal in rhythm and size, and no murmurs were present. The abdomen was protuberant, and shifting dullness was evident. The liver and spleen were not palpable. The genitalia and lower extremities were edematous. There was 2+ pitting in the pretibial area.

The admission laboratory findings were as follows: specific gravity of urine 1.030; pH 6.5; albumin 4+; Esbach 12 Gm.; sugar 2+; and Fantus 3 Gm./L. Fifteen casts and 15 white blood cells per high power field were seen in the urinary sediment. The hemoglobin was 14.5 Gm./100 ml., the white blood cell count was 8,750/cu. mm., the sedimentation rate was 96 mm./hr., and the differential was normal. Total plasma proteins were 4.6 Gm., with an albumin of 2.0 and a globulin of 2.6 Gm. The serum cholesterol was 1,220 mg. per cent. A glucose tolerance test was flat. The antistreptolysin 0 titre was greater than 2,500 Todd units. The serum sodium was 132, the potassium 4.8, the CO_2 21.7, and the chloride 100 mEq./L. The blood urea nitrogen was 19, and the creatinine 2.5 mg. per cent. A urine culture grew alpha hemolytic streptococci.

The patient was given a high-protein, low-sodium diet and chlorothiazide. His weight ranged from 182 to 193 lbs. during the first week. His temperature remained below normal.

Roentgenographic examination of the abdomen showed signs of free fluid. Intravenous pyelograms visualized a faint shadow of the left kidney pelvis. An electrocardiogram was normal. A paracentesis was attempted on two occasions, but only 75 ml. of ascites was obtained. On the eighth hospital day, multiple punctures were made in the legs to drain the edema fluid. The process was repeated two days later. On the fourteenth hospital day, an abdominal paracentesis yielded 2,200 ml. of clear fluid. The patient's weight fell to 169 lbs. At that time, a reddened area and a pustule developed over the lateral aspect of the left ankle. A culture was made and penicillin was started. Later, tetracycline was added. The patient vomited several times a day and seemed to have difficulty swallowing. An examination of the pharynx and esophagus revealed no neurological or obstructive lesion.

After several days of treatment with hot packs and antibiotics, the cellulitis seemed to subside. The patient then steadily improved, and on the thirty-first hospital day his weight was 162 lbs., the cellulitis was healing, his temperature was normal, and the following laboratory data were obtained: serum sodium 132, potassium 4.6, CO_2 6.5, and chloride 104 mEq./L.; blood urea nitrogen 75 and creatinine 4.4 mg. per cent; hemoglobin 9.8 Gm./100 ml.; and white blood cell count 22,400/cu. mm. The urine findings were the same as those secured on admission. Although the organism in the cellulitis proved to be a hemolytic staphylococcus resistant to the antibiotics that were being administered, the patient's improved condition led to the complete discontinuance of antibiotic therapy.

A day later, the patient had a chill, followed by a rise in temperature to 102°F. Examination of the leg wound revealed no increase in inflammation. The chest was dull over the left lower lobe, and bronchial breath sounds were heard. A chest x-ray revealed infiltrates in the right upper lobe and the left lower lobe, and a left pleural effusion. A sputum culture was obtained, and penicillin was

started. In the next 48 hours, the patient voided 6,200 ml. of urine, and his weight dropped to 142 lbs. His temperature rose to 103°F. The urine showed only a trace of albumin, and the Fantus was 4 Gm./L. At the same time, the lungs became more congested, and the sputum became more tenacious. On the thirty-fourth hospital day, while the nurse was aspirating the patient's pharynx, cyanosis developed and respiration stopped.

SUMMARY OF CLINICAL DISCUSSION

Mr. D. R. Wassmuth, junior medical student: The patient had a history of renal disease, and he underwent a nephrectomy at the age of 30. About two years afterward, we are told that the pathologic finding was chronic pyelonephritis. He began to have diffuse edema, and his physician treated him with steroids for a period of nine months. He had complications of a peptic ulcer and cellulitis. The steroids were stopped and the edema progressed.

When he came to University Hospitals, his physical findings were those of diffuse edema, anasarca and ascites. He was afebrile, and he had no physical signs of heart disease. His blood pressure was 150/115 mm. Hg, and the fundi were normal. Laboratory reports showed that he had hypercholesterolemia, hypoproteinemia, massive proteinuria and casts in the urine. The findings of the so-called nephrotic syndrome are principally those that I have recited, with the exception of hypertension, which occurs in what is usually referred to as a "mixed" type of nephrotic syndrome. The other laboratory findings were those that are consistent with this syndrome, except perhaps for the antistreptolysin 0 titre which was quite high and which we can assume from the information given us was possibly due to the cellulitis that the patient had suffered about a month before he came to the hospital.

The urine was cultured for alpha hemolytic streptococci. These are not the organisms that are usually found in chronic pyelonephritis, the standard ones being E. coli and coliform organisms. After extensive urological instrumentation, this disease might be found to have been caused by Aerobacter aerogenes, Proteus vulgaris, Pseudomonas aeruginosa or Staphylococci.

We assume, then, that this patient did not have a pyleonephritis, and we are left with the differential diagnosis of the nephrotic syndrome, the most usual cause of which is glomerulonephritis. Other causes are glomerulosclerosis, amyloidosis and renal vein thrombosis.

We must choose now among those for our diagnosis, and I think that statistically we would have to say that for this patient we must choose glomerulonephritis. Perhaps the attending physicians at this hospital made that diagnosis, for they placed the patient on a type of conservative therapeutic regimen.

He remained afebrile. The edema varied from time to time during the first week. During the second one, various mechanical measures for relieving the edema were tried, apparently with some success. On the fourteenth day of hospitalization, the patient was a little less edematous, perhaps, but his course was basically unchanged. At about the same time, pustules developed over the lateral aspect of the left ankle. The source of infection is problematic, but in any event the lesion was treated and the patient seemed to respond.

Between the fourteenth and thirty-first days, however, he had trouble with swallowing, and some vomiting occurred. These are signs that might be attributed to any of a number of things, but if we were expecting the patient to go into renal failure, we might say that this was a concomitant of uremia. On the thirty-first day, laboratory findings seemed to confirm that viewpoint. The blood urea nitrogen was 75 mg. per cent, the creatinine 4.4 mg. per cent, and the patient was in a severe metabolic acidosis with a CO_2 level of 6.5 mEq./L. We're not told much about his clinical status at that time, except that he seemed to be improving. Yet, the white blood cell count of 22,400/cu. mm. may lead us to doubt that evaluation.

Antibiotics were discontinued, and on the next day the patient developed signs of sepsis and of pneumonitis. X-rays confirmed that he probably had some inflammatory process in his lungs. His pulmonary status became worse. Perhaps he had a pyogenic infection. He died on the thirty-fourth day.

In the long view, we see this patient's course as one of chronic glomerulonephritis, complicated by intercurrent infections and going into uremia with a terminal pneumonitis. We conclude that his course was basically unhindered by the physicians attending him at this hospital, or elsewhere previous to his admission here.

Dr. Paul M. Seebohm, Internal Medicine: The practice of medicine can be a very humbling experience at times.

Dr. R. H. Flocks, Urology: Mr. Wassmuth has given us an excellent discussion, and in many respects I suppose he's right, especially in the last of his statements.

The first point that I should like to discuss a little further has to do with the enuresis. This phenomenon is uncommon after the age of three or four, unless there is an organic reason for it, and one of the reasons for the persistence of enuresis over a long period of time is some type of congenital anomaly in the urinary tract, producing either bladder-neck obstruction or upper urinary tract obstruction. For example, I have a case history here from a recent text on pyelonephritis. The patient was a woman of 24 who had had enuresis since birth. I'll give you the

salient points in it very briefly. When she was 18 years old, hematuria had occurred at intervals for a year, but no adequate studies were made. On examination at a hospital in Boston (Massachusetts General), her blood pressure was 120/80 mm. Hg, soft masses were felt in the abdomen, the urine was concentrated only to a specific gravity of 10.10, pyuria was present, E. coli was cultured, and the retrograde pyelograms demonstrated a pair of huge hydronephrotic kidneys of the type possibly described in our case—congenital hydronephrosis associated with stricture and aberrant vessels at the ureteropelvic junction.

Now in our own experience, congenital hydronephrosis of this type occurs bilaterally in about 80 per cent of cases, although one side may be much more marked or advanced than the other. Thus, one might expect to encounter here, among the findings at postmortem, an underlying congenital ureteropelvic obstruction, and possibly even a bladder-neck obstruction of a mild degree, with an overlying infection or an overlying glomerulonephritis or interstitial pyelonephritis. This is part of the picture of the nephrotic syndrome which was the outstanding clinical impression conveyed to us in the protocol.

The peptic ulcer was probably upon the basis of the steroid therapy that this man had had prior to his admission here. Susceptibility to infection is common with steroid therapy, and also in nephrosis, so that the chances are that the terminal infection may have been upon the basis both of the renal disease and the steroid therapy that the patient had had for the nephrotic syndrome.

The nephrotic syndrome itself was quite typical, as described in the protocol, and many causes for it have been described in the literature. Some of them are as follows. The cause may be renal infection. It may be a toxin such as a mercurial, bismuth or gold. It may be an allergy associated with a serum sickness. The cause may be mechanical, as Mr. Wassmuth has emphasized—renal vein thrombosis, congestive cardiac failure, thrombosis or obstruction of the inferior vena cava. The cause may be a generalized process such as amyloidosis, systemic lupus, or diabetic glomerulosclerosis, or the etiology may be an intrinsic renal disease such as membranous glomerulonephritis or other types of glomerulonephritis associated with tubular degeneration. In some cases described in the literature, the findings in the kidney are very minimal except when examined by means of the electron microscope. That may have been true in this instance. However, the terminal evidence of renal insufficiency makes me think that there must have been a fairly acute pyelonephritis on top of any underlying lesion, which most probably was glomerulonephritis of the membranous type. The immediate cause of death was obviously a staphylococcic pneumonia.

So I should think that there is a good possibility that there was a congenital type of hydronephrosis in this patient, with ureteropelvic obstruction and probably with a superimposed glomerulonephritis. An acute pyelonephritis was probably superimposed upon those conditions as the main source of difficulty, and the terminal event was due to bilateral staphylococcal pneumonia.

Dr. John Keller, Radiology: A film taken of this patient showed the rather homogenous so-called "ground-glass appearance" that we associate with acitic fluid in the abdomen. Otherwise, there was nothing remarkable about the appearance of the abdomen. On another film, a pyelogram, all that was shown, as far as the collecting system was concerned, was a small amount of the renal pelvis and the distal ureter on the left side. A chest film taken shortly before the patient died showed a rather large, dense infiltrate in the right upper lobe, another infiltrate on the left side, and a left pleural effusion. We would interpret these findings as indicating bilateral pneumonia.

Dr. Flocks: I hadn't seen the pyelograms before, and I don't know whether anyone had, but on the basis of it, the kidney pelvis was normal, and it would seem necessary that I retract my diagnosis of congenital renal pelvic obstruction. I'm still not sure that the film was a pyelogram, however.

Mr. R. E. VanScoy, junior medical student: Was there any record of blood pressure taken before the one that is included in the protocol?

Dr. Seebohm: That was the earliest record of a blood pressure measurement.

Mr. VanScoy: I should like to ask Dr. Flocks, then, whether hypertension rules out the use of the term *nephrotic syndrome* in a patient who otherwise seems to have all the symptoms of that condition.

Dr. Flocks: This patient obviously had the nephrotic syndrome. He had all of the characteristics of it. All it is, of course, is a syndrome. It is a clinical picture associated with many etiologies, and the fact that the patient had hypertension makes me feel that he probably had glomerular lesions, since glomerular lesions are associated with high blood pressure. But he definitely had the nephrotic syndrome. The edema, the hypoalbuminemia, the high serum cholesterol—everything fits with it.

Dr. Henry E. Hamilton, Internal Medicine: How do you explain the sudden improvement in the nephrotic state during the terminal phase of the patient's illness, Dr. Flocks? Did the fever play a role there?

Dr. Flocks: You asked me that question several days ago, Dr. Hamilton, and I still don't have a really good answer. The steroids were discontinued because of the acute infection. The steroid therapy did help the edema, and one of the possible explanations that entered my mind is that the patient's adrenals, during the period of termi-

nal stress, may have formed excess steroids that changed the picture. But I don't have a *good* reason.

Dr. F. Tutunji, resident, Internal Medicine: Can pyelonephritis, alone, produce the nephrotic syndrome?

Dr. Flocks: Such cases have been described. Now, I'm sure I have seen hundreds of cases of pyelonephritis, but I have seen very few cases with the nephrotic syndrome. It is quite possible that with the pyelonephritis there must be certain glomerular and tubular changes that are associated with this particular syndrome.

Dr. Jack M. Layton, Pathology: This man had a severe pneumonia, and it was the cause of his death. The entire lower lobes of both his lungs and the middle lobe of the right lung were diffusely involved by severe, confluent, necrotizing and suppurative bronchopneumonia due to hemolytic Staphylococcus aureus. The upper lobes of both lungs also showed patchy involvement by the pneumonic process, so that the patient had suffered a very great reduction in the number of alveoli available for ventilation. Staphylococci were cultivated from lung and spleen tissue, and were demonstrated by means of Gram stains in both. Staphylococci were not demonstrable in kidney tissue by either culture or staining. The left pleural cavity contained 500 ml. of clear yellow fluid, and the abdominal cavity contained about 800 ml. of similar fluid. In the right pleural cavity and pericardial sac, only minimal amounts of fluid were found.

The right kidney was absent, having been removed previously by a surgical operation. The left kidney was greatly enlarged (360 Gm. against a normal of 150 Gm.). It was pale and swollen, with a bulging cut surface and a thickened cortex. Reddish streaks were very prominent all through the kidney cortex. The glomeruli diffusely displayed thick basement membranes, proliferation in glomerular tufts, adhesions and crescent formation. Several tufts were atrophic. Tubules showed secondary degenerative changes. The interstitial tissue had increased in amount, and was edematous and infiltrated with leukocytes. The arterioles were not particularly involved, nor were the small arteries. Thus, terminally, this man did have features of subacute and early chronic glomerulonephritis, but I shall want to say more about this in a few minutes.

The other findings included cerebral edema, septic spleen, fatty metamorphosis of the liver, two small recent erosions in the duodenal mucosa and a very severe ulcerative esophagitis. There was no evidence of any congenital anomaly of the urinary tract at death.

Now I shall return to the kidney for a moment. By light microscopy, there was an increased amount of Schiff-positive material distributed in a patchy fashion along the axial framework of the glomerular tufts. Leukocytic infiltration and epithelial cell proliferation had also occurred in this region. All glomerular lobules were not involved to the same extent. The renal interstitial tissue exhibited edema and cellular infiltrates. The renal tubular epithelium disclosed both atrophic and regenerative changes. The changes in the tubular epithelium were secondary to ischemia and were not indicative of a primary disorder. Arterioles were not appreciably involved.

By electron microscopy, the basement membrane appeared irregularly thickened and folded. Foot processes on epithelial cells were visible, and did not appear to be smudged, flattened or apposed to the basement membrane. Epithelial cell cytoplasm was quite vacuolated. There was a considerable proliferation and swelling of endothelial cells, with abundant highly vacuolated cytoplasm.

This type of change—endothelial-cell proliferation, irregular thickening of basement membrane and relatively little involvement of epithelial cells—is associated with glomerulonephritis.

There are some things here that interest me and that I can't satisfactorily explain. One is that the lesions in the kidney at autopsy were relatively young. I don't believe that the nephritic lesions in the kidney were more than two weeks old. I think it is quite likely that whatever the reason for the nephrotic syndrome may have been, something different was present during the last two weeks of his life. A renal biopsy, taken sometime earlier when he had the nephrotic syndrome, would have been illuminating. By that means, we might have found some type of lesion other than the rather clear glomerulonephritis that he had terminally.

Dr. Flocks gave you quite a list of conditions in which the nephrotic syndrome occurs. I should like to simplify the matter a bit by pointing out that the nephrotic syndrome is associated with lesions in the glomerulus, and that the nature and amounts of protein appearing in the urine may be determined by the alterations in the components of the capillary wall. The lesion may reside in the epithelial cell, which is the common site of involvement in lipoid nephrosis. Some of the cases of lipoid nephrosis that Dr. McCrory and Dr. Kirkendall have sent us from Pediatrics have presented loss of foot processes, smudging of the epithelial foot processes and apposition of the cytoplasm of the epithelial cell to the basement membrane. The change is associated functionally with proteinuria. In some of those children in whom we have had two biopsies—one before and one after prednisone or one of the other corticosteroids was started—the foot processes are shown to be regenerating as the children go into remission, and there seems to be some correlation between the regeneration of the epithelial cell foot processes and the reduction in the pro-

teinuria and remission of the nephrotic syndrome in these cases. Yet we also know from experimental observations that one can selectively damage the epithelial cell, the basement membrane or the endothelial cell predominantly, and that in any of these situations proteinuria may occur. We don't know of any cases studied by electron microscopy in which proteinuria of this degree has occurred without an involvement in the renal glomerular capillary wall. In some of our clinical cases, more than one of these elements have been involved.

I believe that there is a possibility in this case today of an original nephrotic syndrome on a basis other than the anatomical lesion which was found at autopsy. I don't know of a way by which we can make sure of that now, however.

At autopsy, the patient didn't have any lesion in the electron micrographs of the kidney to suggest that he should have had a nephrotic syndrome at the time that he died, and clinically he didn't. It had cleared.

Dr. Seebohm: I suspect that the streptococcal infection prior to the patient's admission would fit into your concept. But we are still left without an explanation for the nephrosis at the onset of his troubles some nine months before admission.

Dr. Layton: I should like to comment on Mr. VanScoy's question about the hypertension that the patient had earlier, when he had a nephrotic syndrome. It is likely that during that particular period he had an endothelial cell lesion too.

I didn't understand Mr. Wassmuth's concluding statement exactly, but it had something to do with the failure of therapy here. I wish to emphasize that this man died from a staphylococcal pneumonia, and not from his renal lesion. Had he survived the staphylococcal infection, I believe that the renal lesion wasn't severe enough to have killed him. In this particular case, we must consider what else might have been done therapeutically to manage the infections that attacked him in the few months before he died.

Dr. Seebohm: I think it might be possible to amplify a little bit on the statements in the protocol concerning the handling of the infection. In reading the notes on the chart, I pieced out the following sequence of events. The patient developed massive edema in spite of all indirect efforts to reduce it. In an effort to combat it, the multiple-puncture method was used in the extremities, and in the course of that therapy, one leg became infected. The pus that accumulated was cultured, and the patient was started on two antibiotics—penicillin and tetracycline. After several days of these antibiotics together with supportive care in the form of hot soaks, the infection cleared. By that time, the infection was known to have been due to a staphylococcus that was resistant to penicillin and tetracycline, but because the wound was healing without the appropriate antibiotic therapy, the attending physicians did not administer Chloromycetin, the drug to which the staphylococcus was sensitive.

When an infection in the lungs appeared two days later, the staphylococcus should have been suspected, and instead of giving penicillin and tetracycline again, the men in charge should have given Chloromycetin or some other antibiotic to which the organism was sensitive, but they didn't.

Student: Isn't it true that penicillin and tetracycline are antagonistic to one another and shouldn't be used together anyway?

Dr. Raymond F. Sheets, Internal Medicine: That effect, I think, has been demonstrated only *in vitro,* and doesn't hold in clinical situations when large doses of antibiotics are involved.

Dr. Seebohm: Yes, but giving these antibiotics together ignores tissue response to infection. The concept as I have understood it is that penicillin is lethal to bacteria at a particular stage of development, and tetracycline is bacteriostatic at a different stage. The organisms held in bacteriostasis by the tetracycline revive after the withdrawal of the antibiotics, and in that sense the two interfere with one another.

Dr. George N. Bedell, Internal Medicine: Even though the microscopic findings in the kidney weren't terribly impressive, Dr. Layton, the kidney was considerably enlarged. How does this make you feel about the duration of the renal disease?

Dr. Layton: The increase in the size of the kidney in this instance was due largely to the accumulation of fluid in the interstitial area. The patient had had a renal disease, obviously, for some time. I believe he had glomerulonephritis for a short time.

Student: To what extent was the increased size of the kidney due to the fact that one had been removed?

Dr. Layton: The nephrectomy had been performed three years earlier. I think that in that length of time the compensatory hypertrophy of the remaining kidney would not have begun to approach this degree.

Dr. Flocks: We don't know how long the hypertrophy actually had been going on in the left kidney because we don't know how badly damaged the right one was at the time it was removed, both congenitally and from the point of view of the chronic pyelonephritis. If it was just a shell of a kidney, as it could have been, the patient may have been born with practically no function on the right side. Thus there could have been quite a marked hypertrophy and a great difference in the functioning of the left kidney.

Dr. Hamilton: Would you expect a man carrying this high a level of cholesterol to have more atherosclerosis than other individuals?

Dr. W. E. Connor, Internal Medicine: I would expect so.

Dr. Layton: The patient didn't have atherosclerosis. Most of the nephrotics who have high degrees of atherosclerosis have also had quite a few intercurrent infections, I believe.

Mr. Wassmuth: I have a question for Dr. Layton. The classical description of glomerulonephritis, from what I can find, is given in Addison's book GLOMERULONEPHRITIS. There, the disease is described as going through a period in which the patient isn't affected much clinically. Then he goes into the nephrotic state for quite some time, and then he goes into the uremic state, in which he loses all of his edema. The proteinuria goes down to a much lower level than that seen in the nephrotic state, and eventually the patient is in the uremic state when he makes his exodus. I should like to know whether kidneys have been investigated at the stage of the disease when it is less florid than in the stage of the nephrotic syndrome.

Dr. Layton: In the end stage of chronic nephritis? Yes, they have been.

Mr. Wassmuth: How do they compare with this patient's findings at death?

Dr. Layton: Kidneys in the end stage of chronic nephritis are usually much reduced in volume, symmetrically contracted and, diffusely, finely granular. Fibrous scarring of the glomeruli is the dominant feature. In the electron micrograph it is very difficult to separate epithelium from endothelium and from basement membrane. They all tend to merge in a scarred mass.

SUMMARY OF NECROPSY FINDINGS

At autopsy, severe, confluent, necrotizing and suppurative bronchopneumonia due to hemolytic Staphyloccus aureus involved the entire lower lobes of both lungs and the middle lobe of the right lung, in addition to patchy foci in the upper lobes of both lungs. Staphylococci were demonstrated by cultivation in lung and spleen, and by tissue staining in lungs. Staphylococci could not be demonstrated in postmortem heart blood or in kidney tissue. The left pleural cavity contained 500 ml. of clear, yellow fluid, and the abdominal cavity contained 800 ml. of similar fluid.

The right kidney was absent, having been removed previously by a surgical operation. The left kidney was enlarged (360 Gm. vs. normal 150 Gm.), and was pale. The capsule stripped easily, and the cut surface was pale and bulging, disclosing a thickened cortex with reddish streaks in it. The glomeruli diffusely displayed thick basement membranes, proliferation in glomerular tufts, adhesions and crescent formation. Several tufts were atrophic. The tubules were dilated, with flattened epithelium, and were decreased in number. The interstitial tissue was increased in amount, was edematous and was infiltrated with leukocytes. The blood vessels were greatly congested.

The other findings included cerebral edema, septic spleen, mild fatty metamorphosis of the liver, two small recent erosions in duodenal mucosa, and ulcerative esophagitis. There were no evidences of congenital anomaly of the urinary tract at the time of examination.

CLINICAL DIAGNOSES

Nephrotic stage of chronic glomerulonephritis. Terminal bronchopneumonia.

STUDENTS' DIAGNOSES

Chronic glomerulonephritis, complicated by intercurrent infections, going into uremia with a terminal pneumonitis.

DISCUSSANT'S DIAGNOSES

Acute pyelonephritis superimposed on glomerulonephritis. Terminal staphylococcic pneumonia.

ANATOMICAL DIAGNOSES

Severe, confluent, necrotizing and suppurative bronchopneumonia due to hemolytic Staphylococcus aureus, bilateral
Glomerulonephritis, subacute and chronic, left
Surgical absence of right kidney
Pleural and peritoneal effusions
Cerebral edema
Ulcerative esophagitis.

Coming Meetings

In State

Dec. 1	**Respiratory Diseases (Iowa Thoracic Society and S.U.I. Department of Internal Medicine).** University Hospitals, Iowa City
Dec. 5-6	**Surgery (Iowa Div. Am. Cancer Soc., and S.U.I. Department of Surgery).** University Hospitals, Iowa City
Jan. 9-10	**Obstetrics and Gynecology (S.U.I. Department of Obstetrics and Gynecology, Division of Maternal and Child Health of the State Department of Health and Iowa Obstetrical and Gynecological Society).** University Hospitals, Iowa City

Out of State

Dec. 1-3	**Surgery of the Hand and Forearm.** University of California, San Francisco
Dec. 2	**Lederle Symposium.** Echo Motor Hotel, Edinburg, Texas
Dec. 2-3	**Low Back Pain.** University of California, Los Angeles
Dec. 2-7	**American Academy of Dermatology and Syphilology.** Palmer House, Chicago
Dec. 4-7	**Advances in Electrocardiography (American College of Physicians).** New York University Medical Center, New York City
Dec. 4-8	**Recent Advances in Diseases of the Chest (American College of Chest Physicians).** Statler-Hilton Hotel, Los Angeles
Dec. 5-8	**Postgraduate Course in Cardiology (Institute for Cardio-Pulmonary Diseases).** Scripps Clinic and Research Foundation, La Jolla, California
Dec. 6-8	**Neuroendocrinology Symposium.** Miami, Florida
Dec. 7-9	**Diseases of the Cornea.** University of California, San Francisco
Dec. 7-9	**New York Academy of Sciences Conference on the Cervix.** Barbizon-Plaza, New York City
Dec. 8-10	**American Psychoanalytic Association.** Biltmore Hotel, New York City
Dec. 9	**Conference on Arthritis.** Presbyterian Medical Center, San Francisco
Dec. 9-10	**Psychiatry in Everyday Practice (University of California, San Francisco).** Stockton State Hospital
Dec. 9-10	**Academy of Psychoanalysis.** Hotel Commodore, New York City
Dec. 10	**Lederle Symposium.** Holiday Inn, Waco, Texas
Dec. 11-22	**Practical Cystoscopy.** Cook County Graduate School of Medicine, Chicago
Dec. 11-22	**General Surgery.** Cook County Graduate School of Medicine, Chicago
Dec. 15-16	**Peripheral Vascular Disease.** University of California, Los Angeles
Dec. 18-22	**Treatment of Varicose Veins.** Cook County Graduate School of Medicine, Chicago
Dec. 18-22	**Proctoscopy and Sigmoidoscopy.** Cook County Graduate School of Medicine, Chicago
Dec. 18-22	**Vaginal Approach to Pelvic Surgery.** Cook County Graduate School of Medicine, Chicago
Dec. 26-31	**American Association for Advancement of Science.** Denver
Jan. 2-6	**Intermediate Electrocardiography for General Physicians and Specialists.** Center for Continuation Study, University of Minnesota, Minneapolis
Jan. 5	**Conference on Proctology.** Presbyterian Medical Center, San Francisco
Jan. 5	**Lederle Symposium.** Admiral Semmes Hotel, Mobile, Alabama
Jan. 7-13	**Eighth Annual General Practice Review (University of Colorado School of Medicine).** University of Colorado Medical Center, Denver
Jan. 8	**Lederle Symposium.** Hotel Lowry, St. Paul
Jan. 12-13	**Cataract Surgery Symposium.** University of Kansas College of Medicine, Kansas City, Kansas
Jan. 13	**Coronary Arteriosclerosis—Detection and Management.** Stanford University, Palo Alto, California
Jan. 13	**Skin Problems in Children.** Children's Hospital, University of California, San Francisco
Jan. 13-14	**Psychiatry in Medical Practice (University of Southern California).** San Bernardino County General Hospital
Jan. 15-18	**Internal Medicine—Today's Problems in Diagnosis and Management, and Tomorrow's Projections (American College of Physicians).** Ochsner Foundation Hospital, New Orleans
Jan. 15-19	**Forensic Pathology.** Armed Forces Institute of Pathology, Washington, D. C.
Jan. 17-19	**Seminar for Aviation Medical Examiners.** University of Kansas College of Medicine, Kansas City, Kansas
Jan. 17-19	**Tenth Postgraduate Course, Diabetes in Review: Clinical Conference (American Diabetes Association in cooperation with University of Michigan Medical School, Wayne State University College of Medicine, Wayne County Medical Society, and Michigan Diabetes Association).** Statler Hilton, Detroit (17 and 19) and University of Michigan, Ann Arbor (18)
Jan. 18-20	**American Society of Clinical Radiology.** Arizona Biltmore Hotel, Phoenix
Jan. 19	**American Society of Facial Plastic Surgery.** Hotel Elysee, New York City
Jan. 19-20	**A Clinic on Human Disability.** Morrison Center for Rehabilitation, University of California, San Francisco
Jan. 20	**Conference on Office Diagnosis.** Presbyterian Medical Center, San Francisco
Jan. 22-24	**First Inter-American Conference on Congenital Defects.** Statler Hotel, Los Angeles
Jan. 22-24	**Clinical Rheumatology.** Mayo Clinic, Rochester, Minnesota
Jan. 23-25	**Obstetric Problems in Private Practice.** Medical College of Georgia, Augusta
Jan. 24	**Lederle Symposium.** Sheraton-Portland Hotel, Portland, Oregon
Jan. 25-27	**Otolaryngology for Specialists.** Center for Continuation Study, University of Minnesota, Minneapolis
Jan. 26	**Nuclear Medicine, Part I begins.** University of Southern California, Los Angeles
Jan. 26-27	**American Society for Surgery of the Hand.** Palmer House, Chicago
Jan. 26-29	**Man and Civilization: Control of the Mind, II.** University of California, San Francisco
Jan. 27-Feb. 1	**American Academy of Orthopaedic Surgeons.** Palmer House, Chicago
Jan. 29-31	**Twenty-sixth Annual Session of the International Medical Assembly of Southwest Texas.** Granada Hotel, San Antonio
Jan. 29-Feb. 1	**Medical Genetics (American College of Physicians).** University of Michigan Medical School, Ann Arbor
Jan. 29-Feb. 1	**American College of Surgeons, Sectional Meeting.** Statler-Hilton and The Biltmore, Los Angeles
Jan. 29-Feb. 3	**Vaginal Approach to Pelvic Surgery.** Cook County Graduate School of Medicine, Chicago
Jan. 29-Feb. 3	**Treatment of Varicose Veins.** Cook County Graduate School of Medicine, Chicago
Jan. 29-Feb. 3	**Proctoscopy and Sigmoidoscopy.** Cook County Graduate School of Medicine, Chicago

Editorials

HOLIDAY GREETINGS

To you and yours we wish a Merry Christmas, and we say, quoting Tiny Tim, "God bless us every one."

Christmas is a happy time, a season of song, of laughter, of tinsel and holly, of gifts and surprises. The youngsters are home from school and college. Children and grandchildren make our holiday replete with joy, though admittedly they create a measure of confusion.

For millions, Christmas possesses a special spiritual significance, as a time of worship, a time to celebrate the birth of the Son of God. For many, Christmas is a season for reflection, for contemplation of one's role in life and of one's place in the scheme of things.

It is an incongruous world in which we live. Medical science has done a great deal to protect and prolong man's life, but it and other branches of science have also been perverted to the wholesale destruction of man and all his works. The continued existence of the human race is dependent upon the whim of a mere mortal, and as never before, the future is in doubt.

This Christmas in the year 1961 is a time when all mankind, regardless of creed or color or nationality, should invoke the Lord to prevail upon men and nations to settle their differences without the cruel instrumentality of war. The catastrophe of nuclear conflict is unthinkable, the effect upon future generations too horrible to contemplate.

Let us all put a fervent prayer for peace and human compassion at the top of our Christmas list.

PULMONARY METASTASIS

We are accustomed to thinking that the development of pulmonary metastases after the surgical removal of a malignancy is ominous, and that little can be done for such a patient other than to keep him as comfortable as possible until death brings blessed relief. A recent report by Moersch and Clagett,* of the Mayo Clinic, affords us a pleasant surprise, however. Of a group of 165 patients upon whom thoracotomy and resection of metastatic pulmonary tumors were performed between 1941 and 1959, an encouragingly large number were benefited.

Eighty-six of the patients with pulmonary metastases were female, and 79 were male, and their ages ranged from four to 75 years, and averaged 50 years. The patients with metastatic cancer averaged 37 years of age. In 37 patients (22 per cent), there were multiple metastatic lesions in the lungs, and in 18 of those, roentgenologic evidence of multicentricity was present prior to thoracotomy. There were 134 patients with metastatic carcinoma of the lung. The primary source of the malignancy was in the rectosigmoid in 36 individuals, the breast in 21, the colon in 16, the kidney in 12, the uterine cervix in 11, and the testes, ovary, thyroid, bladder, etc. in smaller numbers. In 31 patients the pulmonary metastatic tumor was a sarcoma. The primary source of the tumor was fibrosarcoma in 8, chondrosarcoma in 4, osteogenic sarcoma in 5, Ewing's tumor in 2, and rarer types of sarcoma in lesser numbers of patients.

The commonest warning sign of a metastasis to the lung was a chronic cough (37 per cent). The production of sputum occurred in 21 per cent, hemoptysis in 18 per cent, thoracic pain in 13 per cent, dyspnea in 6 per cent, and a respiratory wheeze in 5 per cent of the cases. All of the lesions were visible roentgenologically, and were seen as a nodule or mass shadow of a localized nature. Bronchoscopy and cytological studies were helpful diagnostic aids.

Two-thirds of the patients underwent some type of lobectomy, 21 per cent required pneumonectomy, and only 4 patients required resection of the chest wall or diaphragm as a part of the definitive operation. Of the 165 patients, only four were lost to follow-up studies, and of those four, two were known to have survived eight years, a third was known to have lived six years, and the fourth had survived four years after the operation. The interval that elapsed from pulmonary resection to the last follow-up or to death ranged from less than one month to more than 15 years. Sixty-two (38 per cent) were living at the time of the report—40 per cent of those who had had metastatic cancer were survivors, and 29 per cent of patients with metastatic sarcoma were still living. The absolute one-year survival rate was 71

* Moersch, R. N., and Clagett, O. T.: Pulmonary resection for metastatic tumors of lungs. SURGERY, 50:579-585, (Oct.) 1961.

per cent, and of the 71 patients who were traced and who were eligible for five-year survival studies, 31 per cent were living five years after their operations. The patients surviving three years after operation had a greatly improved prognosis for the succeeding years. Of the patients who had had multiple metastases, there were fewer survivors than of those who had had single lesions, and those with sarcomatous metastases also carried a poorer prognosis. The hospital mortality was 1.2 per cent. Survival was dependent upon the basic growth potential of the tumor.

According to the authors, there are three basic factors which determine the operability of patients with metastasis to the lung. The first requisite is an ability to withstand surgical intervention; the second is adequate definitive treatment of the primary lesion; and the third is the absence of local recurrent lesions or metastases beyond the scope of the projected resection. Obviously, resection is contraindicated in patients with diffuse pulmonary metastases.

The authors conclude: "Thoracotomy and resection of pulmonary metastatic tumors constitute a highly worthwhile and rewarding course in properly selected patients."

PLANTAR WARTS

Plantar warts occur frequently in children and result in considerable discomfort and disability. Numerous methods of therapy have been employed. Curettage of the lesion after the injection of a local anesthetic has been a popular method of treatment, but the injection of an anesthetic into the thick skin of the sole of the foot is painful, and after curettage there usually is considerable discomfort for several days. Roentgen therapy was widely used, but unfortunate after-effects brought this mode of treatment into disrepute. The application of liquid nitrogen has been effective, but frequent recurrences have been reported following that sort of therapy. Occlusive dressings, used with podophyllin, resulted in cures for 60 per cent of the patients after two months of treatment.

Vickers,* in a recent report in the BRITISH MEDICAL JOURNAL, describes the results of a simple method of therapy, formalin foot-soaks for 15 to 20 minutes each night for six to eight weeks. The efficacy of formalin in the treatment of plantar warts was accidentally discovered by Thomson in 1943, when he used a 3 per cent solution of the drug in the treatment of hyperhidrosis. As a result of a survey of 646 children with plantar warts, Vickers found that the formalin soaks resulted in cures of 80 per cent of all plantar warts up to 1.0 cm. in diameter. The larger warts were treated by formalin soaks for three weeks and then curetted. The results, according to the author, are entirely dependent upon the enthusiasm of the doctor in encouraging persistence in the treatment and upon the cooperation of the patient.

The technic of treatment consists in soaking the wart-bearing portion of the sole for 15 to 20 minutes each night for six to eight weeks. Before soaking, the patient is to remove the scale and dead tissue by scraping with the edge of a nail file. Initially, a 3 per cent solution of formalin was employed. If there was little hardening of the skin, the formalin was increased to 5 per cent, 7 per cent and even 10 per cent. Several patients developed interdigital cracks as a result of hardening of the skin, but that could be prevented by the application of petrolatum between the toes before the soaks were started.

The use of formalin therapy is simple, painless, inexpensive and, according to the British author, a very effective method of treatment.

* Vickers, C. F. H.: Treatment of plantar warts in children. BRITISH M. J., 2:743-745, (Sept. 16) 1961.

ULCERATIVE COLITIS

Ulcerative colitis is a serious disease of approximately equal sex incidences. It characteristically occurs in young people, it is chronic in its course, and in its natural history it has exacerbations and remissions that make the evaluation of various therapeutic measures difficult. The malady frequently results in chronic invalidism, and its mortality is considerable, varying from 10 to 20 per cent. Despite a great amount of research, the etiology remains obscure. The disease has been attributed to allergy, to infection, to nutritional causes, and by many, many physicians, to psychosomatic origins.

Treatment is non-specific, and because of the chronicity of the malady, the numerous and serious complications, and the significant emotional component, it is notoriously discouraging to the patient and a constant challenge to the physician. Recent contributions to the management of ulcerative colitis warrant review, for progress is being made, and a measure of hope can be offered to sufferers from this serious disease.

SALICYCLAZOSULFAPYRIDINE

The conventional treatment of chronic ulcerative colitis consists of rest, nutritional repletion, correction of anemia, restoration of electrolyte and fluid balance, control of infection, antispasmodics, and attention to the emotional problems of the patient.[1] In addition to the conventional therapy, various clinicians have reported their experience in the use of additional measures.

In 1959, Moertel and Bargen[2] reported their results from the use of salicyclazosulfapyridine (Azulfidine) in the treatment of 183 patients with ulcerative colitis. In the management of those individuals, they found that 2 per cent were worse,

34 per cent were unimproved, 42 per cent were improved, and remission occurred in 22 per cent. The authors concluded: "The drug is not the final answer to the treatment of chronic ulcerative colitis, but it has been shown to be of considerable usefulness in controlling the symptoms of the disease. Comparison with other commonly employed methods of therapy would seem to indicate that at present, when combined with the basic general and supportive measures, it represents the treatment of choice for chronic ulcerative colitis."

CORTICOSTEROIDS

Kirsner, Palmer and colleagues,[1] of the University of Chicago, in an unusually informative article on the disease, reported a seven-year experience with 240 patients suffering from chronic ulcerative colitis treated with corticotropin and the adrenal steroids. The severity of the disease in the 240 patients was classified as mild in 12, moderate in 86 and severe in 142. Corticotropin (ACTH) or steroid therapy was instituted in those patients not responding consistently to careful treatment in the hospital, in acute fulminating colitis, and in the complications of progressive rheumatoid arthritis, iritis or erythema nodosum. The method of therapy consisted of ACTH alone in 67 patients, various oral steroids in 74, and ACTH followed by oral steroids in 97. The results of therapy were considered good in 114, moderately favorable in 80, and minimal in 45 patients. Therapy, when effective, had a salutary effect upon the clinical picture, on the proctoscopic appearance of the rectum and colon, and on the laboratory tests. Steroids were being continued in 101 patients—over a year in 86, and over two years in 62. Of this group of 101 patients, 89 were leading useful lives. The drug proved to be very helpful in 46, helpful in 48, and ineffective in seven. In 99 patients the steroids were discontinued for varying periods of time, and symptoms recurred periodically in 59 of the group. However, remissions continued in 40 patients—three for less than a year, 31 for over two years, and 12 for over five years. Complications of steroid therapy "excluding the usual hypercorticism" were relatively common. Emotional difficulties were the most troublesome problems in the series. Psychoses developed in 15, three patients committed suicide, and the emotional instability was exaggerated in 22 patients. Infections occurred in 43, hypertension in 45, severe hypercorticism in 35, hyperglycemia and glycosuria in 30, serious electrolyte disturbance in 15, duodenal or gastric ulcer in two, and intestinal perforation in two patients. Death unrelated to the disease or to the steroids occurred in 10 patients, death related to the disease in six, and death attributed to steroids in seven patients. The Chicago group concluded: "ACTH and adrenal steroids, though not curative, are highly effective adjuncts to the total treatment of ulcerative colitis; the effects of the steroids surpass the response to any other therapeutic measure." In this group of 240 patients, only 26 (12 per cent) were referred for surgical treatment.

Watkinson,[3] a British physician, in a meticulously controlled study assessed the value of the rectal installation of topical hydrocortisone hemisuccinate sodium in patients with ulcerative colitis. A comparison was made between the frequencies with which remissions occurred in pairs of patients, one of whom was given potent and the other was given dummy treatments. The author concluded, "The majority of the patients receiving potent hydrocortisone improved and those receiving the inert therapy worsened." Watkinson, in a later report,[4] concluded that use of sulfasalazine is limited because of toxic reactions that develop, and furthermore that remission resulted in but 47 per cent of the 69 attacks that he treated by that means. Oral cortisone administered to 109 patients resulted in remissions in two out of five patients treated, in contrast to the occurrence of remission in one out of six given inert therapy. Topical treatment with steroids in 73 colitis attacks resulted in improvement in 70 per cent, though only 52 per cent showed complete remission. The use of prednisolone suppositories in hemorrhagic proctocolitis was disappointing, for only 44 per cent of 36 patients so treated had temporary remissions.

In another carefully controlled study, Truelove[5] evaluated these agents in trials involving 120 ulcerative colitis patients at Radcliffe Infirmary, Oxford. The patients were divided into three groups of 40 each. The first group were given 5.0 mg. q.d.s. of prednisolone by mouth, and within two weeks one-third of them went into rapid remission. Group 2 received topical water-soluble steroid therapy nightly by rectal drip. Hydrocortisone hemisuccinate and prednisolone-21-phosphate were both used. Nearly three-fourths of the group had rapid clinical remissions. Thus, there was little to choose between the two steroids. The third group of 40 patients were given a combined therapy of oral prednisolone, 5.0 mg. q.d.s., and a nightly drip of either hydrocortisone hemisuccinate or prednisolone-21-phosphate. The results of a two-week treatment were found to depend upon the combination used. Oral prednisolone and prednisolone-21-phosphate rectally had results similar to those achieved by patients receiving topical therapy alone. The patients taking oral prednisolone and receiving hydrocortisone hemisuccinate rectally *all* showed rapid remission, and thus this emerged as the best of the various treatments studied.

The author defined remission as a complete loss of all symptoms within two weeks after the start of treatment, together with a sigmoidoscopic appearance indicating a decisive improvement. For the truly mild attack, Truelove recommended that the initial treatment consist of local steroid therapy

nightly over a period of several weeks. For the minority of mild attacks that fail to abate rapidly following local therapy alone, combined treatment with oral prednisolone should be employed. Those cases that were bad enough to be classified as moderately severe should have the combined oral and local therapy at the outset.

Matts,[6] in an endeavor to simplify the local treatment of ulcerative colitis, provided the patients with disposable plastic enema bags containing saline, to which one tablet of prednisolone-21-phosphate was added. A retention enema of this solution was employed nightly for four weeks. A study of 100 cases treated by Matts showed improvement in 88 per cent, and this improvement was maintained by most of the group for a period of nine months. The patients were provided with reserve supplies of the equipment and the drug, and were advised to resume treatment immediately in the event of a relapse.

SURGERY

Prohaska, Dragstedt and Thompson[7] reported the surgical experience with chronic ulcerative colitis at the University of Chicago. The sustained use of corticosteroids did not inhibit the relentless course of the disease in all patients. Of a total of 387 individuals with ulcerative colitis treated with corticosteroids from April, 1950, to December 31, 1960, surgery was performed upon 81. These were 43 males and 38 females whose ages ranged from 12 to 66 years. The duration of disease in this group of 81 patients varied from two months to 20 years, with the majority coming to surgery within five years of the onset of the disease. Thirty of the patients were considered to be reasonably good risks; 17 of the group were operated upon because of acute, unremitting exacerbations irresponsive to corticosteroids; 34 patients were desperately ill with acute and usually sudden complications.

There were six deaths in the group of 81 patients, all occurring in the latter group of desperately ill patients. There were no deaths in the 19 children who were operated upon. Among the 75 surviving patients, 58 are no longer disabled and are working; 16 patients are partially disabled, and one patient is totally disabled. The Chicago group conclude from their experience that the addition of corticosteroids to the total management of ulcerative colitis does not render the patient intolerant to surgical trauma, nor does it make him unduly sensitive to bacterial infection. They noted only small differences between surgical patients treated with corticosteroids and those not treated in this manner. There were minor differences in wound healing—a definite retardation of wound healing as shown by skin-edge separation when the sutures were removed, by moderate keloid formation, and by a greater incidence of wound disruption. Special care is required in the conversion from corticosteroid therapy to prolonged cortisone protection, meticulous closure of peritoneal surfaces, reinforcement of the abdominal wound closures with tension sutures, and construction of a trouble-free ileostomy.

Goligher,[8] after operations on 145 patients with ulcerative colitis, cautioned against ileo-rectal anastomosis in conjunction with colectomy. He considers it important to distinguish the usual left-sided type from the segmental or so-called right-sided form of the disease in which ileo-rectal anastomosis is perhaps the procedure of choice. For the ordinary diffuse, primarily left-sided colitis, colectomy with ileo-rectal anastomosis inevitably means the retention of diseased rectum and constitutes an invitation to recurrent inflammation or to the development of carcinoma. Of 104 patients subjected to interval surgery, two died, in contrast to 12 deaths in 41 patients who had undergone emergency operations, 12 of whom had perforations. In Goligher's judgment, the higher incidence of perforation in the steroid-treated patients can be attributed to the continuation of conservative therapy, which extended the period during which the patient is a risk, rather than to any deleterious effect of steroids upon the bowel itself.

THE HAZARD OF CARCINOMA

The hazard of carcinomas developing in patients with idiopathic ulcerative colitis has been emphasized by Dennis and Karlson.[9] In a group of 267 patients, carcinoma of the colon or rectum developed in 20, an incidence of 7.4 per cent. A series of 98 patients with the disease were under prolonged observation beyond the tenth anniversary of the onset of the disease, and there had been no evidence of carcinoma at the onset of their illnesses. In 60 of these patients with colons and rectums *in situ* and followed for a total of 218 patient-years, the incidence of carcinoma development was 4 per cent per year. In 54 patients, colectomy had been performed, but the rectums had been retained, and in a total of 229 patient-years' observation of this group, the incidence of carcinoma development was found to be 1 per cent per year. The Minneapolis group concluded that after the tenth anniversary of onset, the patient with idiopathic ulcerative colitis bears an annual risk of cancer development approximately equal to that of procto-colectomy in good hands, quite apart from the other hazards of the disease. After that, the outlook for survival is equivalent to that of a person of similar age without a history of colitis.

Bargen[11] and the group from the Mayo Clinic, in a follow-up study of 1,564 patients with ulcerative colitis, found that fatal carcinoma of the colon developed in 98 patients (6.2 per cent). Carcinoma occurred in patients with ulcerative colitis 30 times more frequently than in individuals of the same age in the general population. How-

ever, the study showed that the typical patient with ulcerative colitis was 31 years of age at the time of his clinic visit, and that he had a better than 50 per cent chance of living 25 years. From their experience, the authors concluded that early removal of the colon was in no way indicated. The annual death rate from carcinoma of the colon or rectum among patients with ulcerative colitis is estimated to be between one and two per hundred, and thus the patient with ulcerative colitis should not be unduly alarmed by the hazard.

Watkinson has stated that the physician who cares for many patients with ulcerative colitis sleeps lightly. It is a disease that tests the character and fortitude of the patient, and challenges the wisdom, the knowledge, the judgment, the skill and the patience of the physician. Progress in the management of ulcerative colitis is being made, but until the precise etiology is clarified and specific therapy is possible, it will continue to tax all the resources of the physician and the surgeon.

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ARE "EQUIVALENTS" REALLY EQUIVALENT?

When laymen talk about medicine, they often speak in terms that everybody can understand, but what they say isn't, to say the least, always accurate. During the Kefauver Subcommittee's investigation of the prescription drug industry, over and over again, it was said that "generic equivalents" will give the American people the same top quality of medicine that is provided today by brand-name drugs. Is that statement true?

Preparations of the same medicine made by different manufacturers are almost certain to be different. Differences in production methods, in the size of particles and in the type of binder or of coating can make marked differences in the drug's effects on the patient.

In a recent edition of JAMA, Levy and Nelson reviewed many studies showing that the difference between brands of the same drug is often therapeutically significant.[1] Boger and Gavin[2] found that supposedly equivalent capsules of tetracycline, made by different manufacturers, produced varied concentrations in the blood because the fillers retarded absorption unequally. Morrison and associates[3] found that the amount of riboflavin absorbed from eight different multivitamin tablets varied from 81 per cent of the labeled amount of riboflavin down to 14 per cent, because of the varying disintegration times of the tablets.

Keller[4] made a study of supposedly equivalent tablets of prednisone made by two companies. He found therapeutic differences between them of such magnitude that when patients on product A were switched to product B, the healing process stopped or was even reversed. Levy and Nelson believe that these differences must be due to variations in the methods of producing the two products.

There is a widespread belief that the therapeutic efficacy of a pharmaceutical product can be determined merely by carrying out the applicable USP assay for the drug content. But this is not necessarily true. Almost all pharmaceutical products contain ingredients other than the active drug, and the manufacture of these products involves a series of operations—mixing, compressing, coating, heating, filtering. Any of these operations, if improperly controlled, can significantly affect the therapeutic performance of the material without the drug assay's showing any deviation in the contents of the drug.

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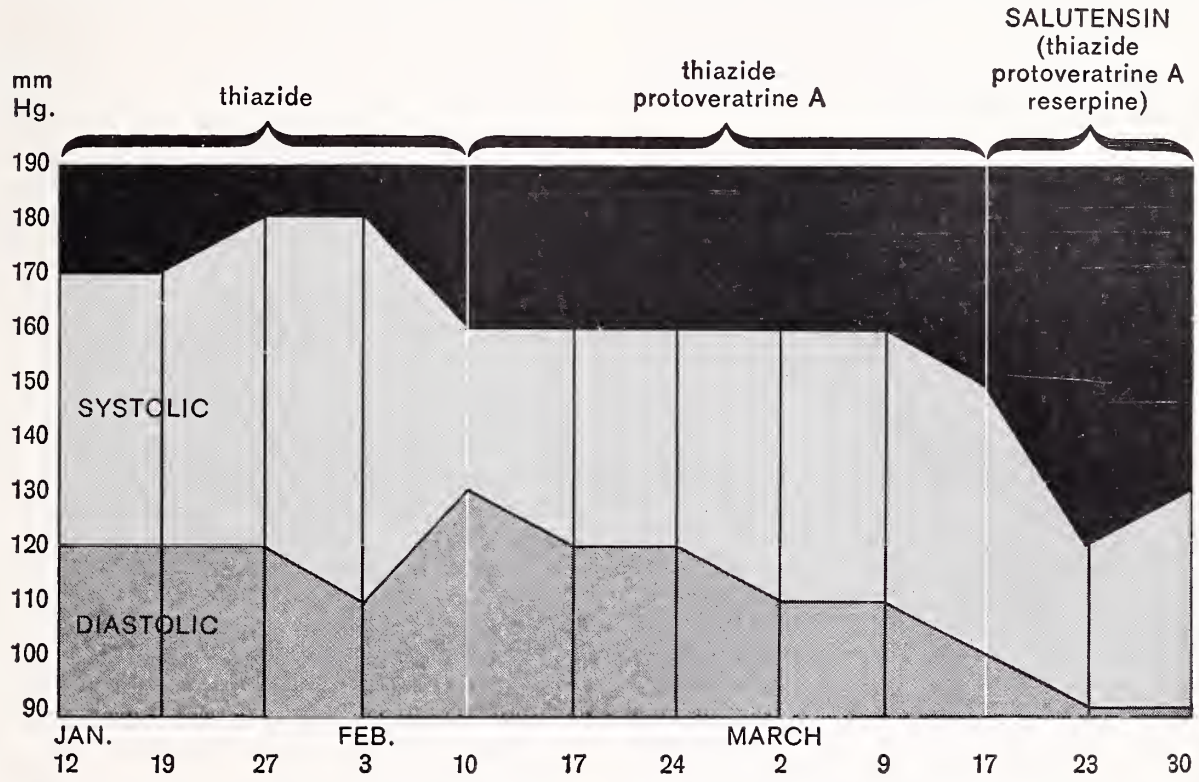
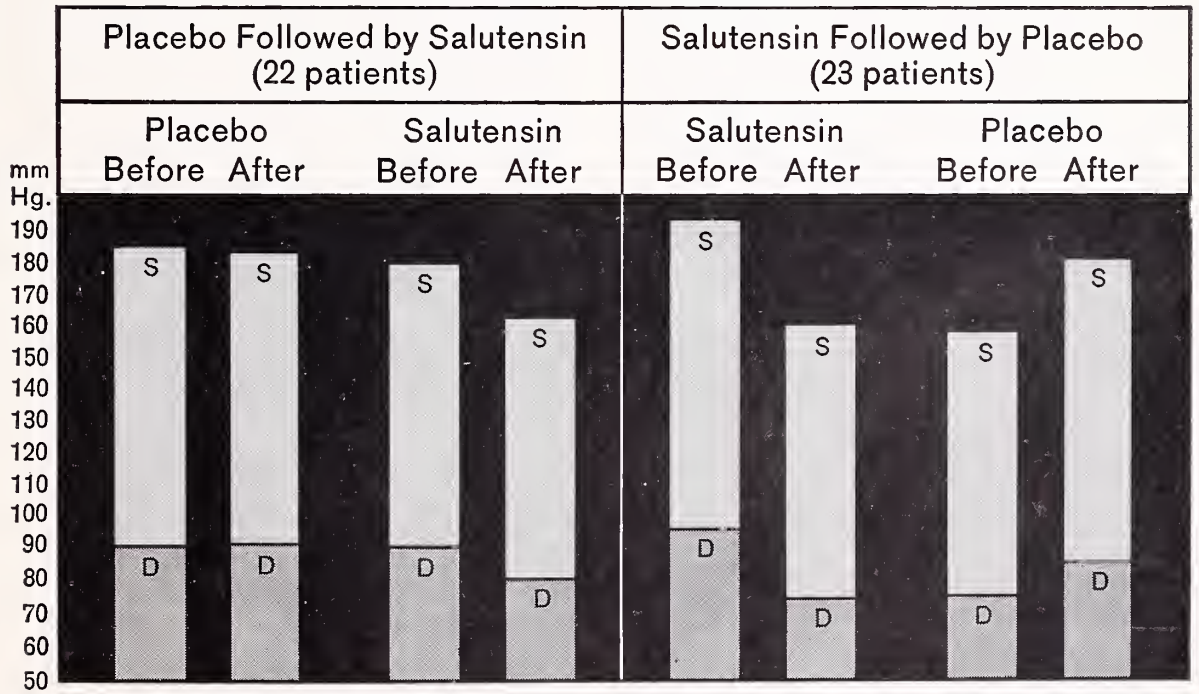
11 WEEKS TO LOWER BLOOD PRESSURE TO DESIRED LEVELS BY SERIAL ADDITION OF THE INGREDIENTS IN SALUTENSIN IN A TEST CASE

(Adapted from Spiotta, E. J.: Report to Department of Clinical Investigation, Bristol Laboratories)



3½ WEEKS TO LOWER BLOOD PRESSURE TO DESIRED LEVELS USING SALUTENSIN FROM THE START OF THERAPY IN A "DOUBLE BLIND" CROSSOVER STUDY

Mean Blood Pressures–Systolic (S) and Diastolic (D)



In this "double blind" crossover study of 45 patients, the mean systolic and diastolic blood pressures were essentially unchanged or rose during placebo administration, and decreased markedly during the 25 days of Salutensin therapy. (Smith, C. W.: Report to Department of Clinical Investigation, Bristol Laboratories.)

DIETARY FAT AND HEART DISEASE

The effect of dietary fat on the level of blood cholesterol can be studied objectively in the laboratory, and the results, if obtained by proper experimentation, are unequivocal. To some extent the effect of the blood cholesterol level on the development of atherosclerosis can be studied in the laboratory, but in direct experimentation, experimental animals must be used, and in many respects these animals do not duplicate accurately the situation in man. In human studies, investigators have had to resort to correlations which obviously have serious shortcomings, but in general the evidence favors a higher incidence of atherosclerosis with elevated levels of blood cholesterol. The all important question that still remains is: What effects do dietary fats have on the atherosclerotic process and the development of coronary heart disease?

Although a preponderance of evidence supports the hypothesis, not all studies are in accord with the idea that excess unsaturated fat in the diet is beneficial. More than an influence on blood cholesterol may be involved, for dietary fatty acids are now known to enter directly into the atherosclerotic plaque, and also these fatty acids have marked effects on certain of the blood-clotting mechanisms. Both of these processes are implicated in the etiology of cardiovascular diseases and do not necessarily conform with current proposals regarding saturated and unsaturated fats.

Evidence has been given that as the severity of atherosclerosis increases, the amount of linoleic acid in the arterial wall increases.[1] The greatest increase has been found in the fatty acids associated with cholesterol in the form of cholesterol esters. If, by theory, linoleic acid is protective against the development of atherosclerosis, why should the atherosclerotic plaque contain more linoleic acid than normal, undiseased tissue?

Several years ago it was shown that peroxidation of the lipids in the arterial wall occurs and as the severity of atherosclerosis increases, so also does the peroxide value of the arterial lipids increase.[2] Peroxidation is to some extent a function of the degree of unsaturation, and one wonders whether or not this phenomenon of peroxidation in atheroma may be a danger signal indicating a potentially harmful result of large ingestion of highly unsaturated fats. Investigators have suggested this possibility and emphasized the need for caution in indiscriminate increases in dietary unsaturated fat.[3]

A great deal has been written about the increasing incidence of coronary heart disease during the past half century. Grouping together all forms of cardiovascular disease as cause of death, the figures given in the STATISTICAL ABSTRACT OF THE UNITED STATES show 287.2 per 100,000 population in 1910 and 515.0 per 100,000 in 1957. There are many factors that should be considered in interpreting such data, but let us assume that these figures do represent a definite increase in the incidence of death due to coronary heart disease during this span of years. In the same interim, the total fat available for consumption in this country has not changed appreciably, although there have been definite changes in the source and characteristics of the dietary fat. Using estimates of the fatty acid composition of dietary fat from 1910 to 1958, it would appear that the total linoleic acid available has increased about twofold during this period. In other words, if total fat and fatty acid composition were the sole factors implicated, the incidence of heart disease should not have increased; if anything there should have been a decrease.

Isolated examples such as these are not presented with the idea of disapproving the hypothesis of a causal relationship between dietary fat and heart disease. There can be cited many good studies that support such a relationship. It is the intent, however, to show why much more information must be obtained before general pronouncements and recommendations are made which will affect the dietary practices of the general public and which may have serious repercussions on agriculture and agriculture-dependent industry.

Lecturers and reviewers have frequently recommended to the public moderation in the consideration of dietary practices. Fat is a major contributor to the total calorie intake, and the excess fat on your serving of roast beef is better for you if left on the plate. A moderate reduction in our total fat intake can result in an important decrease in total calories, certainly a highly desirable goal for most of us. The practice of such moderation may have desirable end results and is nutritionally sound. Unfortunately, a few nutritionists, but nevertheless representative of a very vocal group, advocate a much more involved dietary change for the general public. They would have us eliminate entirely certain fats such as butterfat and replace these fats with liquid vegetable oils. Whether or not this can be accomplished on a practical scale without inadvertently increasing total calories and thus contributing to the problem of obesity has yet to be determined. More serious is the fact that we have no knowledge of the nutritional implications of a major change in dietary practice that would result in the long-continued ingestion of relatively large amounts of highly unsaturated fat. This could be beneficial, but on the other hand, without definite knowledge we cannot be sure that such a change would be completely free from undesirable effects. Certainly such a major change in dietary practice should not be recommended to the general public without further information regarding the long-term implications.

But there is an all important exception that must be made. The physician must apply all avail-

able knowledge in the treatment of a patient who may be in a critical condition. It is the prerogative of the physician to alter the fat intake of his patient in the manner which conforms with his interpretation of the best information available at the time. Furthermore, it is the duty of scientists to make this information available to physicians. Here we arrive at an unfortunate conflict of interests in the dissemination of knowledge resulting in misinterpretations and serious concern among physicians, nutritionists and public health officials, as well as agriculturists. Medical scientists have given information to the public and then have said: "Don't use this information without consulting your doctor." They have given every man a do-it-yourself kit with a label: "Use only under the direction of your physician." An *ad hoc* committee of the American Heart Association has published a report on the relation of dietary fat to heart attacks and strokes.[4] Although published in a medical journal, obviously a major use has been in lay consumption, both through extensive reprint distribution and by excerpting and editorializing in the lay press. There is no major scientific fault in any of the statements that are made in the report, particularly when the summary (added to reprints only) commences with the following disclaimer:

"Question: Should everyone make the changes in the diet described in this report?

"Answer: No one should make significant changes in his diet without medical advice."

Unfortunately, the damage is done. It is only natural for the man in the street to conclude that if a major dietary change is good for the treatment of individuals who have had heart attacks, such a regimen must be good for him also. He has his do-it-yourself kit wrapped up in a very responsible medical journal, and he is going to use it regardless of the "caution" on the label.

How can the public be convinced that medical advice for certain individuals is not advice for the world at large? Medical journals might be condemned for publishing reports for lay consumption, but this is only a small part of the problem, for many media of publication for the layman carry similar information. It is going to be necessary to publicize widely the serious gaps in scientific knowledge that emphasize the ill-advisability of premature recommendations of major changes in dietary practice for the general public. The story cannot be told in completeness today. There is a chance that changes will be advantageous to the public when the facts are known. Effective means must be found to hold the line until that day comes.

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—Richard H. Barnes, Ph.D.
Editorial in the NEW YORK STATE J. MED., **61**:3986-3988, (Dec. 1) 1961.

AMBULATORY TREATMENT OF TUBERCULOSIS

On the basis of experience at some Army hospitals, physical activity *per se* appears not to be harmful to the tuberculous process in the presence of chemotherapy.

Since an abrupt change in physical activity might occur when a patient was transferred from convalescent-leave status to full military duty, it appeared appropriate to test the effect of this added physical stress in the hospital environment. Therefore, a previous program of free ambulation was expanded to include a certain amount of controlled physical exercise, in the form of calisthenics. This was gradually increased to include competitive sports and full-time on-the-job work assignments.

All 105 of the patients had previously untreated pulmonary parenchymal disease. Daily isoniazid and PAS was given was given to all but those unable to tolerate PAS. As a substitute these latter patients received streptomycin. It was also given as an additional drug to some patients with extensive disease.

On admission to the hospital, following initial examinations and the institution of chemotherapy, the patients were started on regular occupational therapy and educational programs. Asymptomatic patients were expected to participate in active calisthenics for 15 minutes per day on a five-day-week basis, beginning within two to four weeks of admission; however, calisthenics were not started in some patients with far-advanced disease and large cavities until two months after admission. The calisthenics were approximately on the level of activity given to regular troops during their basic training period. Rest periods were eliminated for these patients.

When the patients reached the noncommunicable stage, without regard to roentgenographic change, active sports were added, including basketball, volleyball, golf, bowling and swimming. Cavities had usually been resected by that time. A minimum of one hour of active sports a day, five days a week, was required. However, most patients engaged in two hours of sports. When noncommunicable status was reached, on-the-job training was added to the program. The patient was later given a job on the post and was gainfully em-

ployed on a full eight-hour-a-day schedule for several months prior to discharge.

In 62 per cent of the patients, the disease was either moderately or far advanced; 44 per cent had cavitary disease at the time of original diagnosis; 81 per cent had tubercle bacilli in the sputum at the time of original diagnosis.

TREATMENT RESULTS

The results of treatment, as judged by roentgenographic changes, showed that 82 of the patients had either marked or moderate roentgenographic improvement; 19 had slight improvement; and four had no significant roentgenographic change. No patient showed evidence of worsening.

Of the 46 patients with cavitary disease, 20 achieved complete healing within three to eight months on chemotherapy alone. In 24 additional cases, resectional surgery for residual cavities was performed after five to 11 months of treatment. Two patients were eventually discharged with the "open negative" syndrome. No patients with cavities were discharged if their sputum was infectious, and in no instance was there evidence of enlargement of existing cavities or development of new cavitation during the period of observation.

Of the 105 patients, 33 were subjected to thoracotomy—24 for resection of residual cavitation, five for resection of extensive residual nodular disease. In four cases, surgery was for diagnostic purposes.

Reversal of infectiousness occurred rapidly. Seventy-one of the 81 patients with tubercle bacilli in the sputum before treatment were noninfectious by the end of the second month. Only three patients were still discharging tubercle bacilli at the end of the fourth month. One became non-infectious at the end of six months and remained so thereafter. Two patients with noninfectious sputum at the end of two months had a single culture positive for tubercle bacilli at four months, and remained negative thereafter. Both of those patients eventually came to surgery, one at six and one at 10 months. His strain of tubercle bacilli was 100 per cent resistant to isoniazid at that time. At the end of seven months, resectional surgery was performed.

The average duration of hospitalization was approximately 12 months. The patients were discharged at the end of that period and advised to take additional chemotherapy for another six months, making an average of 18 months' total therapy. One hundred of the 105 patients were discharged as fit for military duty. Five were placed on temporary retirement, two of them for administrative reasons. Two of those patients, although unfit for military duty, were capable of living in the general community under reasonably normal conditions, for their limitations were imposed by pulmonary insufficiency rather than pulmonary tuberculosis. Thus, in none of the patients was the tuberculous disease considered a disabling factor at the time of discharge.

The only harmful effects noted in the entire group were due to accidents that had occurred during competitive sports. Two received fractures.

—Abstract of a report by Drs. James A. Wier, James M. Schless, Luke E. O'Connor and Orman L. Weiser in AM. REV. RESPIRATORY DISEASES, July, 1961.

NEW FILMS FOR MEDICAL MEETINGS

Prints of "The Next Step," a 16 mm. black and white sound motion picture dealing with oral polio vaccine and first shown at the New York City meeting of the AMA last summer, can now be borrowed from Pfizer Laboratories, 235 East Forty-second Street, New York City. It presents detailed information on the development, manufacture, testing, immunization properties, and methods used in clinical dispensing of the new live, oral poliovirus vaccine. The film was produced as a professional information service to physicians and allied health workers, and is not to be shown to laymen. Print reservations must be made three weeks in advance, and whenever practical, applicants should list two alternate dates on which they can arrange to show the film. Furthermore, they should mention the size of the expected audience so that printed leaflets can be sent for distribution to those who see the picture.

Physicians who attended the AMA Clinical Meeting in Denver last week saw the premiere of a 16 mm. color-sound film entitled "Cancer Detection: Proctosigmoidoscopy in Office Practice." This 15 min. motion picture covers the necessary equipment for routine proctosigmoidoscopy, home and office preparation of the patient for this procedure, and the examination itself which includes inspection and palpation, proctosigmoidoscopy and withdrawal. Through the use of a special endoscopic camera, the film also depicts the mucosal areas of the sigmoid colon, showing actual pedunculated and nonpedunculated polyps. The film concludes with a diagnostic follow-up with barium enema.

The film can be borrowed without charge from C. B. Fleet Company, Inc., Lynchburg, Virginia, and the firm will provide copies of a 12-page mimeographed booklet for distribution to physicians who see the motion picture.

President's Page

THE DOCTOR'S PART IN CIVIL DEFENSE

Physicians in northeast Iowa participated actively, about a month ago, in planning and presenting a medical civil defense program which attracted delegations from each of the dozen or more counties in that civil defense area, and many of the same speakers who addressed the Oelwein meeting are to discuss their topics again at the December meeting of the Polk County Medical Society, in Des Moines. The details can be found in the "Personals" section of this issue of the JOURNAL.

I urge physicians throughout the remainder of the state to arrange similar medical civil defense meetings, so that the entire medical profession in Iowa can be well informed about the problems that are anticipated, in case a natural or man-made disaster occurs, and about the measures that physicians must take in preparing themselves and their communities.

Shortly, medical self-help training programs are to be set up at the local level, and we shall be called upon to help with them. A committee appointed by Governor Erbe, consisting of Marion E. Alberts, M.D., a Des Moines pediatrician, E. G. Zimmerer, M.D., the state commissioner of health, Paul Johnson, Ph.D., of the State Department of Public Instruction, and a fourth member who will represent the State Civil Defense Office, are to attend a planning session in Battle Creek, Michigan, this month, and subsequently will advise local people in arranging for the classes. It is expected that instructional materials will be distributed in mid-January.

President

THE JOURNAL *Book Shelf*

BOOKS RECEIVED

A MANUAL OF NEUROLOGY AND PSYCHIATRY, by *Ralph T. Collins,* M.D. (New York, Grune & Stratton, Inc., 1961. $6.50).

PSYCHIATRY—BIOLOGICAL AND SOCIAL, by *Ian Gregory,* M.D. (Philadelphia, W. B. Saunders Co., 1961. $10.00).

EYE SYMPTOMS IN BRAIN TUMORS, by *Alfred Huber,* M.D. (St. Louis, The C. V. Mosby Company, 1961. $16.00).

DISTURBANCES OF HEART RATE, RHYTHM AND CONDUCTION, by *Eliot Corday,* M.D. (Philadelphia, W. B. Saunders Co., 1961. $8.50).

PROGRESS IN LIVER DISEASES, ed. by *Hans Popper,* M.D., and *Fenton Schaffner,* M.D. (New York, Grune & Stratton, Inc., 1961. $13.75).

CLINICAL OBSTETRICS, by *Benjamin Tenney,* M.D., and *Brian Little,* M.D. (Philadelphia, W. B. Saunders Co., 1961. $8.50).

DIFFERENTIATION BETWEEN NORMAL AND ABNORMAL IN ELECTROCARDIOGRAPHY, by *Ernst Simonson,* M.D. St. Louis, The C. V. Mosby Company, 1961. $13.50).

SOMATIC STABILITY IN THE NEWLY BORN, A CIBA FOUNDATION SYMPOSIUM, ed. by *G. E. W. Wolstenholme,* M.B., B.Ch., and *Maeve* O'Connor. (Boston, Little, Brown and Co., 1961. $10.00).

BOOK REVIEWS

PHYSIOLOGY OF PREGNANCY, ed. by *Ernest W. Page,* M.D., and ENDOMETRIOSIS, ed. by *Charles S. Stevenson,* M.D. (Vol. 3, No. 2 of CLINICAL OBSTETRICS AND GYNECOLOGY). New York, Paul B. Hoeber, Inc., 1960. $18 per year for four issues).

This volume deals with the very fascinating subject of the physiology of pregnancy. Each facet of maternal and fetal physiology has been quite completely covered by a top man in that particular field. The chapter on lipids is quite technical for the average reader, but it is understandable, and all of the presentations are well written.

The section of the book dealing with endometriosis is a summary of the subject, from etiology to pathology to current surgical and medical treatment. It is a good, well-written review.—*Claude H. Koons,* M.D.

PEDIATRIC GYNECOLOGY, ed. by *John W. Huffman,* M.D., and OBSTETRIC EMERGENCIES, ed. by *Martin L. Stone,* M.D. (Vol. 3, No. 1 of CLINICAL OBSTETRICS AND GYNECOLOGY). (New York, Paul B. Hoeber, Inc., 1960. $18 per year for four issues).

Like its predecessors, this volume is a gem for the reference shelf of the up-to-date Ob-Gyne man or general practitioner. The two broad fields discussed have been covered thoroughly by authors who are authorities in these fields, and upon whose word one can depend.

This series is a must for the physician who wants current information on diagnosis and therapy at his fingertips.—*Claude H. Koons,* M.D.

SURGICAL UROLOGY, SECOND EDITION, by R. H. Flocks, M.D., and *David Culp,* M.D. (Chicago, The Year Book Publishers, Inc., 1961. $10.50).

This book represents a revision of the 1956 edition. It was written primarily for the urology resident, but does constitute an excellent review of all urological surgery in a more or less concise form. It is also very useful for the surgeon who wishes to review a procedure that he does not have occasion to do very often.

The diagrams are good and the text is short and concise. This is an excellent monograph, and the second edition is a distinct improvement over the first.—*Dieter Kirchheim,* M.D.

MEDICAL PHARMACOLOGY: PRINCIPLES AND CONCEPTS, by *Andres Goth,* M.D. (St. Louis, The C. V. Mosby Company, 1961. $11.00).

The author, an M.D., is chairman of the Department of Pharmacology at the University of Texas Southwestern Medical School. With his ambivalent background, he has put together this modest-sized volume designed to give a medical background to pharmacology. He discusses general aspects, neurological drugs, anesthetics, drugs used in cardiovascular diseases, drugs used in gastrointestinal diseases, ones used in endocrine diseases, chemotherapeutic agents, poisons and prescription writing. The type is large and easy to read, the subheadings are boldly silhouetted, and for frosting on the cake, there are many graphs and chemical formulae. Each presentation is intended to be of moderate length in order to convey principles and concepts, rather than minutiae. Consequently, the book isn't too bulky.

In this day and age when we are inundated by myriads of drugs and drug-compounds, it is well for

us to pause and consider some of the pharmacologic fundamentals of the materials with which we are dealing. Each drug salesman comes in with the pitch: "Here we are dealing with an entirely new compound!" We doctors are bewildered and sit in awe, for in our ignorance of fundamentals we can't refute the glib-tongued salesman. If only doctors and medical students would reread a book on the basic sciences every year, they would know a little about these matters and might at least be able to get the detail men to support their claims.

The book is excellently done and eminently practical.—*Daniel A. Glomset,* M.D.

VAGINAL SURGERY, ed. by *Abraham F. Lash,* M.D., Ph.D., and OBSTETRIC ANESTHESIA AND ANALGESIA, ed. by *Robert A. Hingson,* M.D. (New York, Paul B. Hoeber, Inc., 1961. $18 for series of four issues).

Modern methods of anesthesia and improved obstetrical technic have been responsible for the present low rates of maternal mortality (0.38 per 1,000 live births) and infant mortality (2 per cent) in the United States. In underdeveloped countries, the rates range between 2 and 20 per cent for maternal deaths, and between 10 and 50 per cent for infants in the first year of life.

This symposium discusses the types of analgesia and anesthesia in relation to the obstetrical problem at hand, and places adequate emphasis upon the effects on the infant. The dangers in the use of hypnosis are mentioned. There is a fine discussion of resuscitation of the newborn.

The second symposium deals with vaginal surgery, although some of the technics for reconstructive surgery include the abdominal approach. All technics are well illustrated. Simple diagnostic procedures as well as radical vaginal surgery are discussed.

There is also a discussion of "The Medicolegal Aspects of Psychogenic Abortion" included in this volume.—*H. Kirby Shiffler,* M.D.

REHABILITATION OF A CHILD'S EYES, THIRD EDITION, by *Herbert M. Katzin,* M.D., and *Geraldine Wilson,* R.N. (St. Louis, The C. V. Mosby Company, 1961. $3.75).

This book of 107 pages has been written primarily for the parents of cross-eyed children, so that they may understand what happens in cross-eyedness, what steps can be taken in order to correct the condition, what the ophthalmologist wants to do for youngsters so afflicted, etc. I feel, however, that the book is worthwhile also for general practitioners, ophthalmology residents and ophthalmologists, as a review of the subject from etiology right through to the end of treatment.

The book is divided into two parts, the first of them constituting orientation, and the second dealing with rehabilitation. Chapter one discusses the teamwork of the two eyes—their coordination, what makes the eye move, convergence and divergence, fusion and the help it gives in controlling eye movement, and depth perception. Chapter two discusses hyperopia, myopia, and accommodation and its relation with convergence. Chapter three takes up such questions as why eyes cross at birth, at 2½-3 years of age, during or following an illness, and following an injury that has blinded one eye. It also takes up divergency crossing and up-and-down crossing. Chapter four discusses the effect of strabismus on the child.

Under rehabilitation, the author talks, in chapter five, about what the ophthalmologist and the orthoptist do for such patients, and what the parents can do. He also takes up the indications for surgery. Chapter six discusses the treatment of strabismus by means of glasses, and discusses miotics as to esotropia, exotropia and alternating strabismus, and takes up the use of prisms in hyperphoria. Chapter number seven discusses the treatment of amblyopia; number eight, eye exercises; number nine, operations for strabismus; and number ten, treatments after an operation.

There are 24 schematic drawings that make some of the problems easier to understand, and the book ends with a glossary containing definitions of all the terms used in crossed-eye work.

I can recommend this book very highly to all who are interested in working with strabismus. It can be readily understood by all.—*Henry A. Bender,* M.D.

MEDICAL, SURGICAL AND GYNECOLOGICAL COMPLICATIONS OF PREGNANCY, by the staff of Mount Sinai Hospital, ed. by *Alan F. Guttmacher,* M.D., and *Joseph J. Rovinsky,* M.D. (Baltimore, The Williams & Wilkins Company, 1960, $16.50).

This book covers every major complication and most minor disorders that occur during pregnancy, and the authors have done a good job. Each topic has been presented by one or more experts on the staff of Mount Sinai Hospital, New York City, and in most instances a thorough, concise, accurate review has been provided.

The usual disadvantages of multiple authorship—changes in style, etc.—have affected the readability of the book, but are relatively unimportant.

Each of the chapters deals with a general topic such as the cardiovascular-renal system or the pulmonary system, and thus the reader can find what he wants rather quickly, for either quick reference or detailed study. This is an excellent book for any physician who cares for pregnant patients.—*Claude H. Koons,* M.D.

Iowa Association of Medical Assistants

BE PREPARED!

The title of this article, as many of you know, is the motto of the Boy Scouts of America, but it is at least as appropriate for us as it is for the boys in peaked caps and neckerchiefs. The efficient medical assistant must be prepared by keeping her office neat and orderly, and well supplied with the articles that are either regularly or occasionally used there.

Your doctor doesn't attend patients in the reception room, but its appearance can nevertheless affect the success of his practice. It needs clean ashtrays, neat tables or magazine racks stocked with current reading material, and flowers or plants in clean containers well supplied with water. If there are mirrors or windows in the room, they must be free of smudges and finger prints, pictures must be straight on the walls, and the pieces of furniture must be in their proper places on a clean floor.

Examination rooms require different supplies for different types of practice. The daily supply of instruments, syringes and needles should be on hand, sterilized and ready for use. Containers of cotton balls, swabs, dressings and tongue-depressors, and the dispensers of tape, alcohol and germicidal soap should be full each day. Thermometers and other instruments that are kept in a sterilizing solution should be checked each day to make certain that the solution is fresh and that the liquid level is sufficient to cover the instruments. The supply of clean linen should also be checked. Stained or worn towels or drapes should have been put aside for discard or mending at the time the laundry was sorted, so that these won't appear in the examining areas. Open shelves or glass-front cabinets should have plastic-coated paper on the shelves so that they can be wiped off daily with a damp cloth.

The laboratory must be kept scrupulously clean! Into this area materials are brought for testing or for cleaning and sterilizing. Instruments are cleaned immediately after use and set aside in a separate container away from the sterile instruments that are ready for use. Used instruments should be thoroughly scrubbed with a brush, hot water and a soap or detergent, and should be disinfected if necessary before they are placed in the autoclave or sterilizer. Hypodermic needles are checked with air, the cannula of the needle is cleaned with a cotton swab, and the point is checked with dry cotton for barbs before it is sterilized. Using distilled water in the sterilizer not only will prolong the life of that piece of equipment but will keep the instruments bright and new-looking for a longer time. All containers in the laboratory should be clearly marked, and labels should be replaced if they have become stained or blurred. Incidentally, always remember to pour away from the label and to wipe off any drips before replacing the cap or cork and restoring the bottle to its proper place. If supplies are kept in their assigned places at all times, confusion is avoided, but it is still imperative that you read the label *before* using.

If medications are dispensed from your office, your stock should be neatly arranged in whatever order your doctor wishes. Containers must be kept free of dust and should be clearly marked with your cost, the date and amount purchased, and the cost to the patient. Boxes, envelopes, bottles and direction labels should be kept in the same area.

The business office should be efficiently arranged for the person or persons who work there. Equipment should be placed so that no waste motion will be required. Desk tops are kept as free of clutter as possible. What seems organized and neat to you may look like utter chaos to a patient. File cabinets were made to file "in," not "on." Desk and table drawers weren't intended to stand open like bins.

In some offices, an inventory of supplies is kept, a stock book being used for records of the details of each order. By this means, one can tell at a glance the source and price of each item and how soon it should be reordered. Quantity buying is fine if you have sufficient storage space. Reserve stocks should be marked and orders placed *before* supplies have been exhausted. A pocket notebook may be a little bulky to carry around, but it will save steps and time in jotting down notes about needed supplies. Or a want-list kept in the laboratory or dispensary will serve as a reminder of things that must be reordered.

Try to anticipate your needs and order well enough in advance so that a delay in shipment won't cause embarrassment. In the two or three months prior to school opening, you will be needing extra supplies of vaccines for pre-school examinations. Seasonal supplies such as polio and flu vaccines should be ordered well in advance.

Keep your office well supplied and in order. Being prepared spares you the necessity of making excuses or offering apologies.

—Helen G. Hughes

Physicians Throughout Iowa Should Consider Proposing

Homemaker Service for Patients and Their Families

Though it is largely unfamiliar to Iowans, homemaker service is by no means an untried adjunct to health care, for it has been available in various other states for many years—in a few localities since as far back as the 1920's. It was started on a small scale in Waterloo just a year or two ago, it is about to be set up in Polk County, and doctors and other citizens throughout the rest of the state might well start assessing the need for it in their own communities.

The term *homemaker,* in this context, designates a woman 40 or more years of age who has been trained to deal with the special problems that can be found in households containing invalids, and who is employed by a health or welfare agency to perform household tasks and to establish and maintain the atmosphere that is essential to the patient's convalescence. A pleasant personality, physical and mental well-being, experience and a certain amount of training enable such a woman to assume full or partial care of the children in the family, and perhaps of the adults as well. The routine work that she does may include cooking, cleaning, laundering, food buying and providing personal care to the patient and/or the children.

In "the good old days," families afflicted by illness sometimes could call in a relative on short notice to do domestic chores, or such work was done on a turn-and-turn-about basis by neighbors or fellow church members. Whether or not such arrangements actually were common is open to argument, but it is certain that they are infrequent nowadays, and providing stop-gap homemakers has thus become a civic responsibility.

Help of this sort is valuable in myriad situations. At Waterloo, because the homemaker project started under the auspices of the Family Service agency, one of the principal objectives is to preserve unity in families from which the mothers have deserted or have been hospitalized. In a growing number of instances, certainly, women are having to leave their homes to receive treatment for mental disorders, and housekeepers are almost as essential during their absence as after their return. But in Des Moines, where the Polk County Medical Society has been one of the prime movers, help is to be provided only to a family of which a member is ill at home—either the housewife, or the husband or a child for whom the wife can't provide care unaided. Probably no one would argue that a household where the mother is ill needs homemaker service more than does a family that lacks a mother altogether, but the demand for full- or part-time housekeepers is great, and Des Moines and Waterloo have chosen to attempt meeting different segments of it. To fill all such needs completely would necessitate a far more expensive program than can now be undertaken.

ALL HEALTH AND SOCIAL WORK AGENCIES WILL COOPERATE

Virtually every health and social work individual and group in Polk County recognizes the need for a homemaker service and is anxious to cooperate in establishing it and assuring its success. Every physician and every social worker has tried to help sick people either avoid or cope with their problems of daily living, and none of them has been satisfied with the results. Doctors have done their best to secure domestic help for housewives before releasing them from hospitals, and in situations of another kind, have perhaps been unduly

hasty in releasing mothers whose children at home needed care and supervision. Public health nurses have been disturbed about patients whose convalescence seemed impeded by housework and child-care worries. And the Polk County Welfare Department has felt compelled on several occasions to employ untrained and no more than marginally satisfactory housekeepers to care for Old Age Assistance recipients. A time or two, in addition, it has had to provide them for recipients of Aid to Dependent Children. In one such case, the mother was under treatment for cancer and had to have help in caring for her youngsters. Institutionalization was out of the question.

Consequently, 15 or 20 societies, agencies and service clubs have joined together to set up and establish policies for the homemaker service in Des Moines. The Polk County Medical Society has made a sizeable monetary contribution to help get it started, the Auxiliary to the Polk County Medical Society has pledged to it all of the proceeds of its Christmas card sale this year, and doubtless others of the groups are making contributions too.

HOMEMAKERS WILL SUPPLEMENT PREVIOUSLY EXISTING SERVICES

The homemaker service will become an integral part of a health care scheme, the other parts of which have been in operation for many years. The only new workers are to be a social case worker and a stenographer, added to the staff of the Des Moines Council for Social Agencies, and five full-time homemakers (or their equivalent, as will be explained below).

An application has been made to the U. S. Public Health Service for funds with which to pay those individuals' salaries during the first three years of the project, and the local sponsors feel confident that the grants will be made. When the homemaker service has proved itself, it is expected that local funds can be found for continuing it.

Individual physicians will, of course, continue in charge of the medical aspects of their homebound patients' cases, and the public health nurses will continue their visits, providing nursing services in accordance with the attending physicians' instructions and supervising the homemakers in the performance of patient-care tasks that must be done with greater frequency. The director and the assistant director of the Des Moines-Polk County Health Department, both of whom are doctors of medicine, will have charge of the nurses and will serve as liaison between them and the physicians, as in the past.

The new case worker on the staff of the Council of Social Agencies will concern herself principally with deciding which families shall be provided homemaker service and with making sure that the service isn't overutilized in any instance. She will see to it that each family gets the social work services that it needs, but she won't provide those services herself. Rather, they will be rendered, as always, by workers attached to one or another of the public or private agencies in the city. Her further duties will include recruiting and helping to train homemakers, assigning and supervising them, approving payments to them, and in some instances collecting fees for their work.

MANY RECRUITS WILL BE SOUGHT FOR HOMEMAKER TRAINING

The budget submitted with the application for U.S.P.H.S. grants included wages for just five full-time homemakers, but plans are being made to train a considerably larger number of them for work in Polk County. Classes for women interested in the work will be offered through the Adult Education Department of the Des Moines Public Schools. A one-semester course consisting of 10 two-hour sessions is to be presented twice each year, and it is hoped that enough recruits for two simultaneous classes of 15 each can be enrolled each term. Those who complete the course and pass the final examination will be eligible for service as homemakers, and if everything goes well, a lengthy roster will be built up.

The training program may seem a bit ambitious for a service that is expected to employ just five full-time homemakers, but several factors require that women trained and available for such work be quite numerous. An effort will need to be made to match the personalities of homemaker and family members; simple geography will frequently be important; and each homemaker can be expected to voice preferences as regards hours of work and perhaps other details.

Doubtless it also should be pointed out that the employment of just five full-time homemakers or their equivalent in part-time workers doesn't mean that only five families can be served at any one time. Because some families will need no more than a half-day of homemaker service per week, it will be possible for each homemaker to serve two or more families simultaneously.

Fees are to be collected from the families served, graduated according to their abilities to pay, and when homemaker service is provided to a family that is receiving monetary assistance from a social work agency, the agency providing it will be charged a fee approximating the actual cost of the homemaker service.

Elsewhere in the country, homemaker services have in some instances been abandoned for lack of funds, and no one thinks that such schemes can ever be operated at a profit. But they save money for hospitals and health-insurance organizations, thus preventing at least some rate increases, and they confer immeasurable benefits of intangible sorts upon sick people and their dependents.

The experiments at Waterloo and Des Moines thus deserve careful watching.

THE DOCTOR'S BUSINESS

Things You Should Tell Your Insurance Company

HOWARD D. BAKER
WATERLOO

Everyone who buys equipment realizes that it will need periodic inspections and adjustments if it is to continue giving complete satisfaction, but by no means everyone is aware that life insurance policies require periodic attention. One can't just put such documents away in a safety deposit box and forget them. As the years pass, all sorts of events can take place to alter the policyholder's original plans, and an insurance company can't do a thing to adjust his policies to the changes that have occurred in his situation. He must take the initiative himself.

Here are seven sorts of events that may require changes in your life insurance. When any of them affects you, make sure to seek competent advice and then notify your insurance companies of the changes you want made in your policies.

1. Change of Address. Moves from one locality to another are some of the main causes for lapsed policies and lost policyholders.

2. Repayments of Loans. If one borrows from a bank and uses his life insurance policies as security, he assigns his rights under those policies, and there is little doubt that the insurance companies are notified of that assignment. But when the loan has been repaid, the policyholder must make sure that a properly executed release is filed with each of the insurance companies.

3. Marriage or Divorce. A policyholder who marries or becomes divorced undoubtedly will want to change the beneficiary provisions in his insurance policies. Until he makes such changes, however, the company or companies are bound by contract to pay the person previously named. A great deal of financial hardship and ill will has been caused by failure to make such changes.

Mr. Baker is a partner in Professional Management Midwest, and manager of its Retirement Planning Department. He majored in accounting and business administration at S.U.I., and was an agent of the U. S. Bureau of Internal Revenue for 3½ years before forming his present association in 1953.

4. Birth or Death of a Dependent. When a child has been born to the policyholder or when a beneficiary has died, a revision in beneficiary provisions may be necessary, lest the insurance proceeds be paid contrary to the policyholder's wishes.

5. Need for Curtailing Premium Payments. If one finds it necessary to curtail his expenditures for life insurance, he should get his insurance agent to assist him in finding the most advantageous way to do it. Rights upon surrender or lapse differ greatly from policy to policy, and one must seek full information about those rights.

6. Desirability of a Change in the Disposition of Dividends. Generally, the policyholder has a choice among (a) leaving dividends to accumulate interest, (b) using them to reduce annual premiums, and (c) taking them in cash. Furthermore, he usually can change from one of those arrangements to another at will. But unless he makes his wishes known to the company, it will continue the disposition settled upon at the time the original contract was made.

7. Change in Financial Situation. The policyholder's financial status may change. He may have increased his income substantially, he may have inherited some money or he may have mortgaged his home or some commercial property. Any of these events will call for his taking a new look at his life insurance program.

A major shift in the source of his income may have put him under Social Security or, perhaps, may have eliminated him from such coverage. Acquiring or losing eligibility for a group insurance or pension plan may have made comparable changes in the estate that he can leave to his dependents.

Any of these occurrences will require increases or facilitate reductions in your life insurance program, and the companies can't initiate such altera-

tions. Just as in the case of equipment, life insurance needs periodic attention, and the real responsibility for giving it that attention is yours.

SMALL INVESTORS IN BIG REAL ESTATE

John C. O'Byrne*

The interest of the small investor in big real estate continues to grow. The small investor has learned through isolated and individual transactions of his own and through the current publicity that has been given to larger scale development corporations that investment in prime commercial property may provide spectacular growth and some special income tax advantages.

Several factors combine to make commercial real estate attractive. First, the investor hopes to ride the inflationary trend through increase in the value of the property. Second, a major part of the cost can be financed with borrowed money. Third, many excellent firms prefer to rent rather than own and are prepared to execute long-term leases for prime properties. Fourth, deductibility of depreciation and interest on loans may postpone or reduce the realization of taxable income.

Let's say that a corporation develops a $300,000 property. It might put up $100,000 of its own money and borrow the rest. Insurance companies and other institutional investors have been willing to provide mortgage money for sound commercial developments. Their security is not only the structure but the assurance of a long-term lease with a desirable tenant. Using the rents to pay off the loan, the corporation ultimately obtains a $300,000 property for its initial investment of $100,000.

Now add the effects of an inflationary trend. If inflation averaged 4 per cent a year for five years, not an unreasonable estimate, the property would be worth $360,000 at the end of five years. If the corporation sells the property for $360,000 and pays off the loan, it has a gain of $60,000—not on a $300,000 property, but on its $100,000 initial investment.

In effect, the combination of inflation in real estate values and the fixed obligation has allocated to the initial investment all of the growth factor. If the initial investment can be reduced and the borrowed capital increased, the picture is even brighter.

The tax handling of real estate transactions has had much to do with their investment popularity. There is nothing sacred about the corporation. All real estate holdings are entitled to the same treatment. The major item of tax advantage is the deduction for depreciation. Depreciation is allowed as a tax expense even though it is not a cash outlay. In addition, depreciation is figured on the total cost price of the structure, including the full amount of the mortgage. Although depreciation theoretically makes up for wear and tear, and for decline in value, depreciation may be taken as a tax deduction even though the value of the property is actually increasing.

In other investments such as blue chip stocks or bonds, the total cash return is usually taxable income. But in the case of rental real estate, the taxable income may be less than the cash income because of the non-cash depreciation deduction.

In balancing out his cash return and his taxable income, the real estate investor has:

1. The deduction for interest which is both cash outlay and cash deduction
2. The payments on the principal of the loan, which constitute cash outlay but are not deductible
3. Depreciation which involves no current cash outlay but affords a tax deduction.

The real estate investor hopes to come out ahead with "non-taxed" return, or to permit the structure to pay for itself through "non-taxed" rents. In the longer run, he wants to realize his investment growth as capital gains on the ultimate sale of the property.

When the investment vehicle is a corporation, the same tax notions apply, but there is an additional step to get the corporate income into the hands of the shareholders. Distributions by a corporation to its shareholders are ordinary dividend income to the shareholders to the extent that the corporation has income. Thus, if the real estate investment of the corporation produces cash return, but not taxable income, distributions to the shareholders are not taxable dividends. Any distribution made by a corporation which is not out of corporate earnings is regarded as a return of capital to the shareholder. This reduces the cost or basis of the shareholder's stock by the amount of the distribution. This, of course, results in no tax. When the non-dividend distributions from a corporation have equalled the amount of the shareholder's original investment in the corporation, any additional capital distributions are treated as capital gains, just like capital gains on the sale of stock.

The notion of the pooled investment in real estate is adapted to the needs of the small investor. His lack of know-how is supplied by the corporate managers. In addition, the cautiousness of quality, long-term tenants and the conservativeness of institutional lenders provide a little more assurance to the small investor that the managers of the corporation will not lead him astray.

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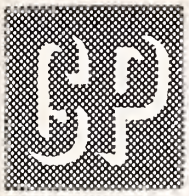
* Mr. O'Byrne is director of the S.U.I. Agricultural Law Center, and this article originally appeared in his column, "Agri-Legal Briefs," in THE AGRICULTURAL BANKER, September, 1961.



Iowa Chapter of the American Academy of General Practice

ADVANTAGES OF MEMBERSHIP IN THE AAGP

The first advantage of membership in the American Academy of General Practice is that it identifies the physician with the aims of the group. Those aims or purposes, as listed in the Constitution, are as follows.

1. "To promote and maintain the highest standard of general practice." The word *maintain* sets the aims of the Academy apart from those of any other medical group, for it recognizes the undeniable fact that no doctor *ever* completes his quest for the highest standard. Each active member is required to attend at least 150 hours of lectures during each three-year period, of which at least one third must have been at lectures sponsored by the Academy or by a medical school. For private study, the Academy publishes a monthly journal which is meant primarily for the family physician and, therefore, covers the entire field of medicine.

2. "To encourage medical students to become qualified family doctors." In Iowa there has been good cooperation between the S.U.I. College of Medicine and the Academy, and each spring during the postgraduate refresher course, in Iowa City, one or more of the classes of medical students are guests of the Academy at a dinner. Moreover, representatives of the Academy have given lectures to the students each year, and most of the preceptors are members of the Academy. The good relations between the Academy and the medical school can grow as the problems of student scholarships, graduate education, research and faculty improvement are dealt with. A new two-year general practice residency may soon become standard throughout the country.

3. "To preserve the general practitioner's right to practice medicine to the full extent of his ability." This aim has drawn fire from several self-centered groups on the basis of restrictions that have been imposed upon general practitioners, however unjustifiedly, in some areas. Yet, through the establishment of general-practice sections in hospitals and through representation on key committees, the wholesale restriction of surgical privileges to those with special membership certificates has been slowed down in most places.

4. "To provide postgraduate training opportunities for the family doctor." The Iowa Chapter alone has planned and presented about 60 hours of lectures during the past 12-month period. The American Academy of General Practice was the first organization to plan such training programs systematically throughout the country.

5. "To promote the science and art of medicine and surgery and the betterment of the nation's health and welfare, and to preserve the right of free choice of physician to the patient." The Academy has stayed out of politics, but the great concern of each member is that freedom of medicine be maintained. The burden of proof is on the critics to point out a better system somewhere else in the world. A strong and united organization such as the American Academy of General Practice will help prevent the encroachment of politicians upon the private-enterprise system of health care.

FRINGE BENEFITS

There are also many fringe benefits of Academy membership. First of all, each member automatically receives copies of the magazine GP and of the HAWKEYE GP NEWS, which is edited in Des Moines. Second, postgraduate courses are open to members at reduced registration fees or none at all. Many fine opportunities are offered during each year for members to enlarge and enrich their practices through listening to lectures. Third, the group insurance program is growing rapidly, and at present includes eight plans: (1) group disability, which includes health and accident; (2) family hospital and nurse; (3) practice overhead; (4) accidental death or dismemberment; (5) supplemental income, providing up to $500 per month for the first two years of disability; (6) group life plan, on the "professional liability plus" arrangement; (7) family catastrophic hospital and nurse expense, which may now be carried for life rather than just to age 70; and (8) a combination of conventional endowment policy and mutual-fund investment plan under which the beneficiaries (whether the doctor and his wife, following his retirement, or the doctor's widow and children) will be assured of receiving a fixed basic income plus a variable amount depending upon the price for which the mutual-fund shares can then be sold. Of this last type of insurance, either the annuity

portion or the mutual-fund portion, or both, can be purchased by Academy members in most states, including Iowa.

If you are not now a member of the Iowa Chapter of the American Academy of General Practice and wish further information, please write to our office in the Bankers Trust Building, Des Moines 9, or visit it at your next opportunity.

IMPARTIAL MEDICAL TESTIMONY—IS THERE SUCH A THING?

There can be no such thing as impartial or nonpartisan medical testimony, according to an editorial in the October issue of TRAUMA, a medical journal for lawyers. Mr. Marshall Houts, the editor-in-chief, says: "By labelling the medical witness 'impartial,' we do not alter his basic attitudes which stem from a lifetime of professional practice. Some doctors see a protruded intervertebral disc at the drop of a hat as the cause of most low back pain. Others scoff at this disease entity and admit only grudgingly that it can exist. Some readily state that myocardial infarction can result from a blow to the chest while others are reluctant to accept this etiology."

Mr. Houts takes issue with the AMA House of Delegates which unanimously endorsed the concept of nonpartisan medical testimony at its last annual meeting. Although the AMA concept is founded on the premise that partiality can be based only on monetary considerations, Mr. Houts feels: "It omits the other potent factors which are always involved. Most medical opinion is based on factors which are purely subjective with the doctor. The grading of an ankle jerk as I, II, III or IV involves subjective determinations in the mind of the doctor. The same is true with classifications of 'slight,' 'moderate' or 'severe' in the measurement of gross atrophy or in interpreting 85 per cent of all x-ray studies." According to him, "The most dangerous witness of all is the witness who labors under the illusion that he is totally and completely impartial and nonpartisan."

Mr. Houts thinks that the answer to the problem lies in orienting all doctors to the courtroom and to the intricacies of the adversary system.

CLINICAL RHEUMATOLOGY

The Mayo Clinic and the Mayo Foundation will present a postgraduate course on "Clinical Rheumatology" on January 22, 23 and 24, in Mann Hall, Medical Sciences Building, Rochester, Minnesota.

The registration fee will be $5 and the tuition $55. The number of physicians who can be accommodated is limited, and those who wish to attend should communicate with M. G. Brataas, secretary for postgraduate courses. The American Academy of General Practice will give credit to those of its members who attend.

CANCER SCREENING FOR BOBBYSOXERS

Women of all ages should be screened for cervical cancer, according to an article by Dr. J. H. Ferguson, of Miami, in the October 28 issue of J.A.M.A. No age limit should be imposed, he says, on the application of smear examinations as a means of detecting early cancer of the cervix.

Among 1,500 women from whom positive cancer smears were obtained at the University of Miami-Jackson Memorial Hospital, 77 were under 20 years of age, Dr. Ferguson reports. The youngest was 14 years of age. Screening examinations, generally, have been restricted to women 30-35 years of age or older, the group in which there is the highest prevalence of cervical cancer.

With universal application, according to an accompanying J.A.M.A. editorial, the screening method has "the potential of eradicating" cervical cancer.

TREATMENT OF DIABETES

A Statement by the Subcommittee on the Teaching of Diabetes in Hospitals, of the Committee on Professional Education, American Diabetes Association

Because of its prevalence and chronicity, diabetes mellitus should be the continuing concern of all physicians, regardless of their type of practice. An essential part of treating the condition is teaching the patient how to live with it.

As in any educational program, a systematic approach should be used. Each physician should have certain specific objectives clearly in mind as he teaches his diabetic patients.

To aid him, the American Diabetes Association has prepared the following check list of nine basic elements of treatment, which constitutes a minimum program for diabetes management. There are many other aspects of treatment which are not mentioned, but they are not so important as are the following:

1. Diet
2. Urine testing
3. Action of insulin and other hypoglycemic agents
4. Technic of insulin injection and sites for it
5. Care of syringe and of insulin
6. Symptoms of hypoglycemia
7. Symptoms of uncontrolled diabetes
8. Care of the feet
9. What to do in case of acute complications.

This guide is not only of value in the initial education of a new diabetic, but can also be most helpful to both patient and physician in the subsequent years of management.

STATE DEPARTMENT OF HEALTH

Edward D. Zimmer
COMMISSIONER

DIABETES MELLITUS

Diabetes, the eighth leading cause of death in Iowa during the year 1960, is a chronic condition that has consistently appeared as one of the ten leading causes of death. It has fluctuated between seventh and ninth because of the variations in death rates for such conditions as influenza and pneumonia, or the variation in numbers of deaths from conditions peculiar to infancy, both of which are more responsive to the factors that lead to epidemoid patterns in statistical tabulations.

As can be seen from Table 1, Iowa has generally exhibited a higher rate of loss from diabetes than has the nation as a whole, primarily because of the differences in age distribution of the populations. However, the differences are not so great as to prevent our getting a reasonably valid idea of what conditions in Iowa must be by interpolating the data that relate to the United States as a whole. The data presented in the following discussion were obtained from the U. S. Public Health Service, and constitute a sample designed to represent the total national experience.

Good therapeutic control measures have radically increased the life expectancy for diabetics, until now it is approaching the life expectancy of the general population. The emphasis on familial case finding, the increased control of infections through the use of drugs and antibiotics, our increased knowledge of vascular system problems, and our greater recourse to dietetic and/or medical aid—all of these have helped to achieve this happy result. Only about 8 per cent of the known diabetics are not under medical care, and almost all of those have received medical advice at some time. National Health Service data indicate that when interviewed 90 per cent of all admitted diabetics denied any chronic limitation of activity, and 82 per cent of those over 65 years of age did not feel themselves to be chronically limited in activity.

If conditions in Iowa can be regarded as reasonably similar to those in the United States as a whole, we can apply the rates obtained by the National Survey to the Iowa population. Thus, we should expect to find approximately 25,000 known diabetics in Iowa, of whom about 2,000 are not presently under medical direction. However, the prevalence rates obtained in the National Survey included only those cases in which the family had been informed of the diagnosis. In addition, the family had to believe that the case had been active

TABLE 1

DEATHS FROM DIABETES MELLITUS

Year	No. of Deaths		Population in Thousands		Rates Per 100,000 Population		
	U. S.[1]	Iowa[2]	U. S.[3]	Iowa[4]	U. S.	Iowa	Difference
1950	24,419	462	151,683	2,621	16.1	17.6	1.5
1951	25,047	443	154,360	2,619	16.2	16.9	0.7
1952	25,474	444	157,028	2,628	16.2	16.9	0.7
1953	25,796	433	159,636	2,635	16.2	16.4	0.2
1954	25,151	400	162,417	2,629	15.5	15.2	–0.3
1955	25,488	448	165,270	2,682	15.4	16.7	1.3
1956	26,184	422	168,176	2,716	15.6	15.5	–0.1
1957	27,180	448	171,198	2,739	15.9	16.4	0.5
1958	27,501	488	174,060	2,729	15.8	17.9	2.1
1959	28,160[5]	467	177,261	2,743	15.9	17.0	1.1
1960	—	482	—	2,759	—	17.5	—

TABLE 2

DEATHS FROM DIABETES DISTRIBUTED BY AGE GROUP
IOWA—1960

Age Group	Frequency		Per Cent		Population[6]		Rates Per 100,000 Pop.	
	Male	Female	Male	Female	Male	Female	Male	Female
0-4	—	—	—	—	156,766	150,448	—	—
5-14	—	2	—	0.7	281,373	269,265	—	0.7
15-24	2	3	1.1	1.0	174,759	183,516	1.1	1.6
25-34	5	3	2.6	1.0	156,081	160,775	3.2	1.9
35-44	6	2	3.1	0.7	164,745	170,360	3.6	1.2
45-54	14	17	7.3	5.8	150,235	152,268	9.3	11.2
55-64	25	36	13.1	12.4	125,847	133,414	19.9	27.0
65+	139	228	72.8	78.4	149,241	178,444	93.1	127.8
TOTAL	191	291	39.6	60.4	1,359,047	1,398,490	14.1	20.8

within the year prior to the time of the interview. It is the opinion of some who have evaluated the results of mass screening programs that the number of undetected cases of diabetes may equal the number of known cases.

Like many other chronic conditions, diabetes appears to a greater extent among elderly people. For the U. S. civilian non-institutionalized population, the prevalence rates are as shown in Table 3.

TABLE 3

INCIDENCE OF DIABETES MELLITUS IN THE UNITED STATES[8]

Age Group	Per Thousand Males	Per Thousand Females
0-24	1.1	0.7
25-44	4.9	3.9
45-54	11.2	13.7
55-64	25.2	31.5
65-74	34.4	50.3
75 plus	31.5	38.8

The relatively higher rates for women at older ages are consistent with the picture obtained from mortality data for Iowa.

A slightly higher number of known diabetics have been found by the survey to live in rural areas. The prevalence rates per 1,000 population were found to be 24.8 for urban, 26.9 for rural non-farm, and 27.1 for farm residents.[7] However, the differences don't appear to be statistically significant at the 95 per cent level of confidence. Possible bias in selection, if it exists, does not appear to penalize the rural groups, since the number of urban residents interviewed was almost twice that of the two rural groups combined.

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INFLUENZA IMMUNIZATIONS

Recent bulletins from the Surgeon General of the U. S. Public Health Service have indicated the possibility of an increase in influenza during the coming winter. The statement was made, "We are probably due for some Asian influenza outbreaks, since they come in two- to three-year cycles." The bulletins also drew our attention to the possibility of extension of the outbreak of influenza in England during early 1960 to the United States during the coming winter.

Dr. Albert P. McKee, director of the WHO Regional Influenza Laboratory at Iowa City, reported to us on October 5, 1961, that the Asian strain of influenza virus had been isolated from material obtained from a patient in Clinton County. California has reported the isolation of the same organism, as has one of the states on the Eastern Seaboard.

A very definite interest in influenza immunization has been expressed in Iowa. The questions most frequently asked of this Department are, "Who should be immunized?" and "What is the recommended dosage schedule?" The following

paragraphs have been prepared as answers to those questions.

The Public Health Service and other groups interested in influenza control especially recommend the immunization of the so-called "high risk" groups of persons. It is further recommended that these groups be immunized with polyvalent vaccine as soon as possible in the fall, and no later than the start of the usual influenza season in December. It must be remembered that a two-week period is required following immunizations before the development of a protective level of antibodies can be expected.

Basically, there are few changes from 1960 in the recommended dosage schedule. There is stronger emphasis on the necessity of two injections of the vaccine for persons who had no influenza immunizations the previous year. The 1960 high-risk categories of patients are again used. They include:

A. Patients of any age who suffer from chronic, debilitating diseases such as:

1. Rheumatic heart disease, especially mitral stenosis
2. Other cardiovascular diseases such as arteriosclerotic heart disease and hypertension
3. Chronic bronchopulmonary disease such as asthma
4. Diabetes or Addison's disease.

B. Pregnant women.

C. All persons over 65 years of age.

It is further recommended that in years of high expected incidence, persons in the medical and health services, public utilities, public safety and education protect themselves by immunizations.

The dosage schedule is as follows:

A. Persons who had no immunizations against influenza last year:

1. Adults (13 yrs. of age and older) 1 cc. subcutaneously, followed by a second injection of 1 cc. from two weeks to two months after the first injection
2. Children (6 to 12 yrs. of age) 0.5 cc. doses administered at the times stated above
3. Children less than 6 yrs. of age, an initial dose of 0.1 to 0.2 cc. Since the dosage suggested is smaller because of the possibility of febrile reactions, it is well to reduce the interval between the two injections to one or two weeks.

On this basis, many are suggesting a third or booster injection of the same strength a couple of months later.

B. Persons immunized last year with influenza vaccine need have only one injection of the dosages listed above. The booster injection two to three months after the first should be administered to small children.

IOWA HAS RELATIVELY FEW NEW TB CASES

Figures released early in November showed that Iowa ranked forty-seventh among the 50 states in new tuberculosis cases reported per 100,000 population last year, but Dr. Edward T. Blomquist, chief of the Public Health Service's TB program is far from satisfied with the record of the nation as a whole.

Data that he has just received from 50 states and the District of Columbia put the number of new active cases reported during 1960 at 55,494—a rate of almost 31 per 100,000. Although that represented a five per cent decline from 1959, Dr. Blomquist said it is below the rate considered necessary if tuberculosis is to be eliminated as a major public health problem.

For the years 1952 to 1959, the annual rate of decline in the new-case rate averaged 8 per cent. Public health authorities say that with presently available means of control, the rate of decline should be 10 per cent. "We do not expect an upsurge in tuberculosis in this country," Dr. Blomquist said, "but the problem has reached the stage where the decline must be accelerated or the prevalence of disease will level off, and tuberculosis will drag out as a long-term, expensive public health problem." The direct cost of tuberculosis to the nation is currently $700,000,000 per year.

NEW ACTIVE TUBERCULOSIS CASES BY STATE, 1960

(With rates per 100,000 population and the rank order)

State	Newly Reported Active Cases		
	Number	Rate	Rank Order
United States	55,494	30.8	
Cont. U. S.	54,977	30.7	
Alabama	1,302	39.8	12
Alaska	224	98.2	1
Arizona	713	54.1	3
Arkansas	811	45.4	6
California	5,129	32.4	17
Colorado	308	17.5	40
Connecticut	377	14.8	45
Delaware	142	31.6	19
Dist. of Columbia	511	67.1	2
Florida	1,418	28.4	23
Georgia	1,151	29.1	22
Hawaii	293	45.6	5
Idaho	58	8.6	50
Illinois	4,063	40.2	11
Indiana	1,195	25.6	29
Iowa	354	12.8	47
Kansas	202	9.3	49
Kentucky	1,138	37.3	13
Louisiana	1,033	31.6	20
Maine	167	17.1	42

State	Newly Reported Active Cases Number	Rate	Rank Order
Maryland	1,407	45.2	7
Massachusetts	1,363	26.4	28
Michigan	2,597	33.1	14
Minnesota	518	15.1	44
Mississippi	716	32.8	15
Missouri	1,144	26.4	27
Montana	154	22.7	36
Nebraska	198	14.0	46
Nevada	68	23.6	33
New Hampshire	72	11.8	48
New Jersey	1,651	27.1	26
New Mexico	460	48.0	4
New York	6,807	40.5	10
North Carolina	1,088	23.8	32
North Dakota	109	17.2	41
Ohio	2,714	27.9	24
Oklahoma	542	23.2	34
Oregon	443	25.0	30
Pennsylvania	3,707	32.7	16
Rhode Island	196	22.9	35
South Carolina	726	30.4	21
South Dakota	122	17.9	39
Tennessee	1,454	40.7	9
Texas	2,640	27.5	25
Utah	71	7.9	51
Vermont	83	21.2	37
Virginia	1,668	41.9	8
Washington	713	24.9	31
West Virginia	600	32.3	18
Wisconsin	821	20.7	38
Wyoming	53	16.0	43
Puerto Rico	1,938	82.2	

CROP-SPRAY CONTAINERS POSE A POISON HAZARD

Empty pesticide containers pose a health hazard in agricultural areas, according to an article in the November issue of ARCHIVES OF ENVIRONMENTAL HEALTH. The major difficulty appears to be associated with exposure of workers to smoke from the burning of paper bags that have contained the poisons, and with exposure of children to residuals left in glass or metal containers.

These hazards were reported by a group of men at the USPHS Communicable Disease Center in Wenatchee, Washington, and the documentation consisted of accident reports in the orchards of the Pacific Northwest, but comparable problems could arise in parts of the country where other sorts of dusting and spraying are common.

The smoke from burning pesticide bags contains a significantly higher amount of the poison than does the air during the spraying operation, and workers should be warned to avoid breathing it. As for the glass or metal cans and jugs, they said that farmers have reused such containers after only one desultory rinsing, or none at all.

MORBIDITY REPORT FOR MONTH OF OCTOBER 1961

Disease	1961 Oct.	1961 Sep.	1960 Oct.	Most Cases Reported From These Counties
Diphtheria	0	1	0	
Scarlet fever	156	95	150	Johnson, Polk, Woodbury
Typhoid fever	0	1	3	
Smallpox	0	0	0	
Measles	31	23	55	Polk, Woodbury
Whooping cough	14	4	22	O'Brien, Polk
Brucellosis	6	11	26	Linn
Chickenpox	61	28	163	Buena Vista, Polk, Scott
Meningococcic meningitis	2	0	1	Dubuque, Woodbury
Mumps	94	35	196	Dickinson, Polk, Story
Poliomyelitis	0	4	0	
Infectious hepatitis	120	139	38	Boone, Polk, Pottawattamie, Scott, Wapello
Rabies in animals	19	32	26	Chickasaw, Story, Woodbury
Malaria	0	0	0	
Psittacosis	0	0	0	
Q fever	0	0	0	
Tuberculosis	23	24	62	For the state
Syphilis	82	109	146	For the state
Gonorrhea	109	156	192	For the state
Histoplasmosis	0	4	1	
Food intoxication	0	0	22	
Meningitis (type unspecified)	13	34	1	Clay
Diphtheria carrier	0	0	0	
Aseptic meningitis	0	13	3	
Salmonellosis	1	1	2	Polk
Tetanus	0	0	0	
Chancroid	0	0	0	
Encephalitis (type unspecified)	0	1	3	
H. influenzal meningitis	1	0	0	Cerro Gordo
Amebiasis	0	3	1	
Shigellosis	1	3	8	Lee
Influenza	14	10	211	Clay

Woman's Auxiliary News

OUR PRESIDENT SAYS—

As a worthy and appropriate message to all of you for December, I wish to offer the following lines from "Keeping Christmas," by Peter Marshall:

"In a world that seems not only to be changing, but even to be dissolving, there are some tens of millions of us who want Christmas to be the same —with the same old greeting 'Merry Christmas' and no other. We long for the abiding love among men of good will which the season brings—believing in this ancient miracle of Christmas with its softening, sweetening influence to tug at our heart strings once again. We want to hold on to the old customs and traditions because they strengthen our family ties, bind us to our friends, make us one with all mankind for whom the Child was born, and bring us each again to God who gave his only begotten Son, that whosoever believeth in Him should not perish, but have everlasting life. So we will not 'spend' Christmas . . . nor 'observe' Christmas. . . . We will 'keep' Christmas—keep it as it is in all the loveliness of its ancient traditions. May we keep it in our hearts, that we may be kept in its hope."

—Mrs. Benjamin F. Kilgore
President

NEW AUXILIARIES ORGANIZED IN 1961

The Woman's Auxiliary to the Iowa Medical Society gladly and sincerely welcomes two newly organized Auxiliaries.

Dickinson County: Plans were begun in the spring for the organization of this Auxiliary. After a meeting with the organization committee, this new Auxiliary began to function in September with the following officers: president—Mrs. R. J. Coble, Lake Park, and secretary-treasurer—Mrs. D. S. Farago, Arnolds Park.

Marion County: The Marion County Auxiliary was organized on September 1, 1961. The following officers were elected for the coming year: president—Mrs. D. A. Mater, Knoxville; vice-president—Mrs. D. H. Hake, Knoxville; secretary —Mrs. A. W. Byrnes, Knoxville; and treasurer—Mrs. F. P. Ralston, Knoxville. Both of these new organizations have appointed committee chairmen and are taking an active part in the Auxiliary's programs in their area.

RURAL HEALTH AND THE THREE C'S

Rural Health is the newest area of interest for our Woman's Auxiliary here in Iowa, and it is quite a recent interest for the National Auxiliary, too. We have entered it at the instigation of the AMA Rural Health Council, which feels that we, as well as the AMA and the state medical societies, should assist farm groups to help themselves in promoting good health for farm people.

Research in the area of rural health has brought out several interesting facts:

1. There are approximately 37 zoonotic diseases —ailments transmissible from animal to man, and hence a concern to rural residents.

2. Farming is second only to heavy construction in the frequency of occupational accidents, principally because of the modern farmer's using increasingly heavy and dangerous machinery.

3. Farm people are often the victims of allergies, for they are close to pollens and are constantly exposed to dust in their fields and barns.

4. More and more potent fungicides and insecticides are being used, and there are accompanying dangers to those who use them carelessly or in too heavy concentrations.

5. With increasing frequency, hormones and antibiotics are being used in feeding and caring for livestock. The possible side-effects to human consumers of meat, milk, etc. are still unknown.

Thus, for these specific reasons, it is apparent that rural health is something to which doctors should devote special attention, and in which the Auxiliary can help. Your co-chairmen of Rural Health have asked that each Auxiliary president appoint a rural health chairman, and have sent a letter containing general instructions that they assume she has given to her rural health chairman. That letter contains a list of packaged programs, pamphlets, skits and films for use in helping rural groups that are having programming problems.

As we think together about the ways in which we can work most effectively, let's keep in mind the three C's that our national chairman, Mrs. Chenault, has suggested: Communication, Cooperation and Coordination.

How do we communicate? We establish contacts with rural women's organizations (Ag. Extension, Farm Bureau, 4-H, to name a few), and let them know that the county Auxiliary is anxious to

cooperate with them in every possible way, and has pamphlets, skits, films and perhaps speakers available for their programs. We should ask them to tell us of ways in which we can be helpful.

In particular, just now, we should inform farm women's groups about the AMA Regional Conference on Rural Health that is to be held at the Savery Hotel, in Des Moines, on May 18-19, 1962, and encourage as many as possible of them to attend it.

How do we cooperate? We Auxiliary members should cooperate locally in such activities as the following:

1. Assuring the success of county, state and regional rural health conferences.
2. Physician placement in rural areas.
3. County fair exhibits.
4. Health fairs in rural areas.
5. Science fairs.
6. Health education programs (in P.T.A.'s, Farm Bureaus, etc.).

How do we coordinate? We coordinate already established Auxiliary projects, such as health careers recruitment, community service, safety and civil defense, with our efforts to work with farm women's organizations.

1. We can look for opportunities to sponsor health-careers programs in rural high schools and 4-H clubs. Perhaps we can offer to transport rural students to hospitals and medical centers for open houses, etc.

2. We can provide programs and information on proper nutrition, and disseminate information on the health problems of the particular rural area.

3. We can urge the establishment of driver education courses, provide information on how to minimize dangers in the handling of poisonous substances, and encourage implement dealers to set up safety instruction programs.

4. As part of our contribution to Civil Defense, we can sponsor courses in first aid, home nursing, etc. and can help kindle enthusiasm for the self-help courses that will be started next month.

It is obvious, we are sure, that our opportunities to be helpful will be varied and numerous. Our first year will be largely a pioneering one, for we must first find out where we can be of greatest service to these groups. Let's show our interest and friendliness—invite farm women to our meetings, and ask permission to attend theirs. Let's make their acquaintance as soon as possible, and offer our assistance in whatever they are trying to do in the health field.

Rural people, like doctors and doctors' wives, place a high value upon the American principle of private enterprise. It is not their custom to beat the drum for a welfare state in this wonderful land of ours. They are our kind of people! Let's work with them, help them and get to know them better.

The time to move into our new program of the three C's is NOW!

—MILDRED LEINBACH (MRS. S. P.)
—SHIRLEY BUXTON (MRS. O. C.)
Co-Chairman, Rural Health Committee

COUNTY AUXILIARIES

Wapello

The Woman's Auxiliary to the Wapello County Medical Society met at the home of Mrs. John Stewart, November 7. Following a report of the district meeting at Knoxville, our local nurses training program and the Future Nurses' activities were reviewed. Mrs. Burn Bannister gave an interesting history of politics in Ottumwa. Local election returns were received during the social hour.

Mrs. Justus Roberts and Mrs. Edward Ebinger were the assisting hostesses.

Wright

The Wright County Auxiliary held its fall meeting at the Club Cafe, Eagle Grove, following a dessert luncheon.

Mrs. Dale Harding, president, conducted the business meeting, and reports were given by members on the projects that the Auxiliary sponsors: Rural Health, Operation Coffee Cup, Mental Health and Future Nurses Clubs.

"Operation Coffee Cup" has been the organization's number-one project, and each of the members has made use of the Ronald Reagan record before various groups and in several homes. The packaged materials were displayed at the meeting and were taken home by several members.

A skit on "Civil Defense" was presented by three members, and a silent white elephant auction raised a substantial amount for A.M.E.F.

WELCOME NEW MEMBERS-AT-LARGE

Mrs. G. I. Armitage, Osceola (Clarke)
Mrs. J. R. Doran, Ames (Story)
Mrs. J. M. Gacusana, Akron (Plymouth)
Mrs. R. H. Heise, Story City (Story)
Mrs. C. N. Hyatt, Corydon (Wayne)
Mrs. C. E. Sampson, Creston (Union)
Mrs. G. H. Scanlon, Iowa City (Johnson)
Mrs. J. A. Sibley, Ames (Story)
Mrs. J. L. Smith, Ames (Story)
Mrs. H. E. Stroy, Osceola (Clarke)

DISTRICT MEETINGS

The eleventh annual meeting of the Medical Auxiliary of District III was held at Vern and Coila's Club, in Arnolds Park, on October 4. The Sioux County Auxiliary members were the official hostesses for the meeting, and members from Clay, Lyon, O'Brien, Osceola, and newly-organized Dickinson Counties attended. Mrs. Keith Swanson, of Hull, president of the Sioux County Auxiliary, welcomed the group, and Mrs. James Thomas, of Sibley, gave the response. Mrs. R. J. Hassebroek, of Orange City, led in a pledge to the flag. Reports and correspondence containing general comments were presented by spokesmen for the various Auxiliaries, and made the morning business session most interesting.

The District's medical representative on the board of the Iowa Division of the American Cancer Society, Dr. E. B. Getty, of Primghar, was the guest speaker at the morning session. He explained all the phases of the Iowa Division's work and acquainted everyone with its achievements. His remarks were timely and significant, since this is the year when the General Federation of Women's Clubs is urging all its members to have uterine examinations. The Iowa Division of the American Cancer Society gave $110,000 to S.U.I. this past year for research, and gave lesser amounts to Iowa State University, at Ames, and Drake University for the same purpose. Helping to finance cancer research is one of the most valuable projects of that health agency.

At the luncheon, in the Bamboo Room, an autumn atmosphere had been carried out in the table decorations. Dr. Dean King, the IMS District Councilor, was a luncheon guest and spoke on the current activities of the Iowa Medical Society. Mrs. E. M. Honke, state chairman of Community Service, spoke on the work of her committee. Mrs. Dean King, of Spencer, chairman of the Committee on Legislation, stressed the importance of writing and talking to our congressmen about the King-Anderson Bill, which is to be one of the measures that the present administration will urge upon the next session of Congress. The Ronald Reagan record "Operation Coffee Cup" was enjoyed by everyone, and provided valuable information for the members to take back to their respective communities.

—MRS. LESTER R. HEGG
Councilor, District III

The members of the Marion County Auxiliary acted as hostesses at the Ninth District meeting, in Knoxville on October 18. A noon luncheon was served at the Maple Buffet, with a fall theme of fruits and flowers carried out in the table decorations.

The meeting was called to order by Mrs. L. F.

Among those in attendance at the District IX meeting were: Front row, left to right: Mrs. A. C. Richmond, Fort Madison, president-elect; Mrs. F. L. Poepsel, West Point, recording secretary; Mrs. J. H. Matheson, Des Moines, treasurer; Mrs. L. F. Catterson, Oskaloosa, councilor, District IX; Mrs. Hazel T. Lammey, Des Moines, administrative secretary; Mrs. D. C. Wirtz, Des Moines, chairman of the Nominating Committee.

Back row, left to right: Mrs. D. A. Mater, Knoxville, president of the Marion County Auxiliary; Mrs. G. S. Atkinson, Oskaloosa, chairman BULLETIN Committee; Mrs. B. F. Kilgore, Des Moines, state president; Mrs. K. M. Lemon, Oskaloosa, president of the Mahaska County Auxiliary; Mrs. C. N. Melampy, Ottumwa, president of the Wapello County Auxiliary; Mrs. E. A. Vorisek, Des Moines, finance chairman.

Catterson, of Oskaloosa, the Ninth District Councilor. She introduced the state officers, and they responded with reports of their respective plans and accomplishments. Reports of county Auxiliary activities and projects were then given by the county presidents. Mrs. B. F. Kilgore, of Des Moines, the state president of the Woman's Auxiliary, explained the duties and program of the state organization.

The thirty members attending were from Des Moines, Ottumwa, Oskaloosa, Corydon, Centerville, Fort Madison, West Point, Pella and Knoxville.

—MRS. THOS. D. CLARK

THE IMPORTANCE OF CIVIL DEFENSE

The objective of the program of Civil Defense is to teach people how to survive a national emergency and meet their own health needs. To attain this program, the people should search for information from a reliable source and then study it carefully. The public is more conscious than it has been of the requirements needed for survival, and public apathy is disappearing fast. This objective should prevail throughout all communities, without mass panic, confusion or hysteria. Soon, "self-help" programs will be available, whether for dealing with the consequences of a natural disaster, or for surviving a military attack.

Following any disaster, emphasis centers upon emergency medical care. A well-organized Civil Defense program in your community, with the medical profession, the local Auxiliary, and the general public cooperating in making preparations, will increase the numbers of people who can be rescued. Let's assist our local Civil Defense organizations, both through the Auxiliary and through the other organizations to which we, as individuals, belong.

Preparedness is the key to survival. Don't panic . . . Prepare! Find out what is needed, and then find out how to meet the need.

The "self-help" training program has been developed, at the national level, by the U. S. Public Health Service and the Office of Civil and Defense Mobilization in cooperation with the AMA Council on National Security and Committee on Disaster Medical Care. In mid-December the members of a committee appointed by Governor Erbe will attend a conference in Battle Creek, Michigan, and shortly afterward they will begin distributing instructional materials and will arrange courses in "self-help" in towns throughout Iowa. The classes probably will start in mid-January. Governor Erbe's committee consists of Dr. Marion Alberts, a Des Moines pediatrician representing the Iowa Medical Society, Dr. E. G. Zimmerer, the state commissioner of health, Dr. Paul Johnson, of the State Department of Public Instruction, and a representative of the state Civil Defense office.

—MRS. SIDNEY BRODY, *Chairman*
Civil Defense Committee

AMEF

American Medical Education Foundation goals for the year are: Every County a fund-raising event for AMEF; Every County a program on AMEF; Every member to learn of, to work for, to give to AMEF.

"Windmill" stationery is still available at 50 cents per packet and the "Corn" pattern at $1.00 per packet. Your AMEF chairman will give you full information on the AMEF playing cards at $2.00 per double deck. Also plan to use the Sympathy, Utility and Appreciation Cards as memorials. It is important that all checks be made out the AMEF-Auxiliary Fund *but* send the checks to me lest Iowa not receive credit for the contribution.

Remember much needs to be done to strengthen our medical schools. I'm expecting much "fan mail" with checks enclosed.

MRS. LEO PEARLMAN, AMEF Chairman
3715 River Oaks Drive
Des Moines 12, Iowa

NEW STATE OFFICERS NAMED

Following the resignation of Mrs. E. B. Dawson as president-elect, the Board of Directors chose Mrs. A. C. Richmond, Fort Madison, to fill the unexpired portion of the term. Mrs. C. A. Trueblood, Indianola, was elected to fill the unexpired term of first vice-president, a vacancy created by the resignation of Mrs. Richmond, and Mrs. Ivan Sayre, St. Charles, was elected to fill the unexpired term of Councilor, District X caused by the resignation of Mrs. Trueblood.

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